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Cholera, its protean

Cholera.

Vol. 1.

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By Dr. G. Archie Stockwell, F.Z.S.

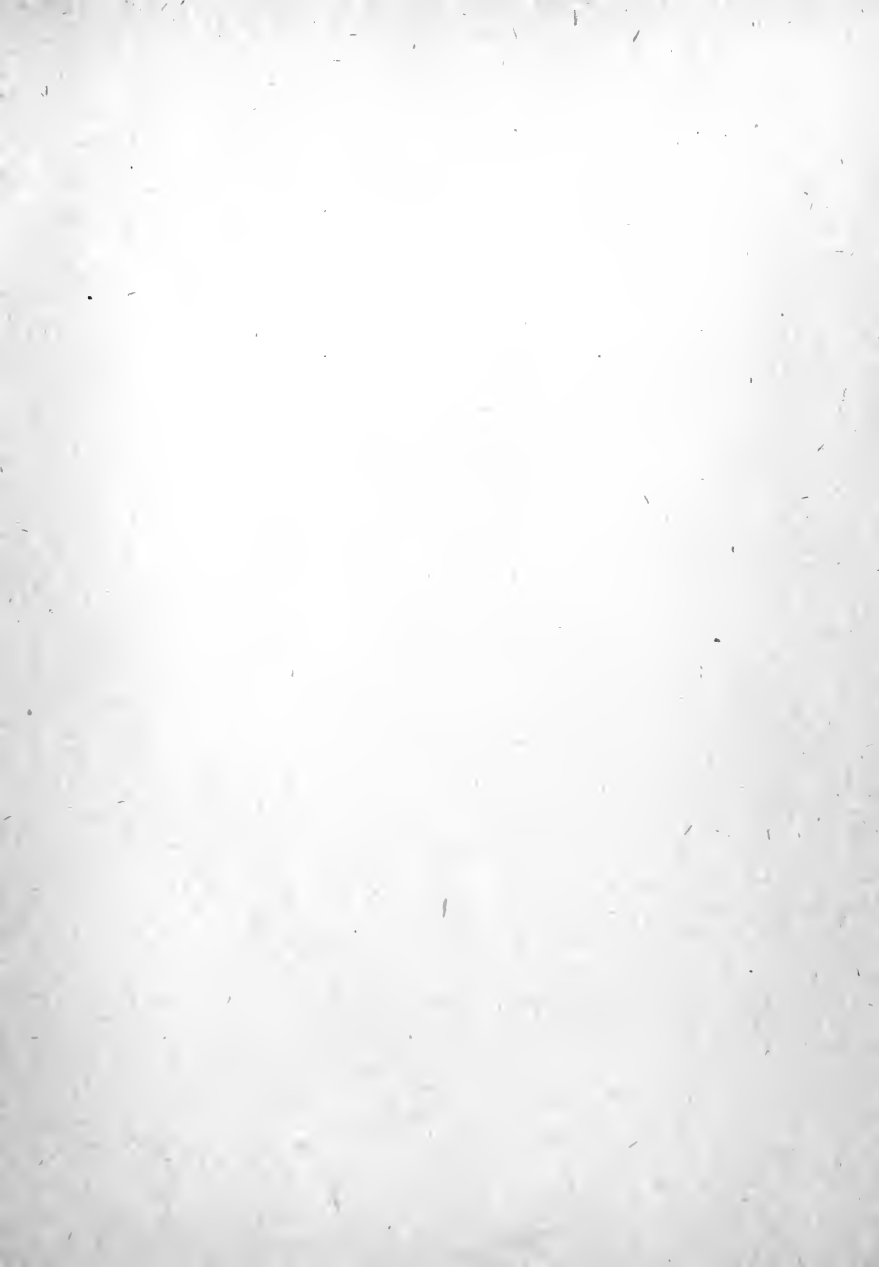
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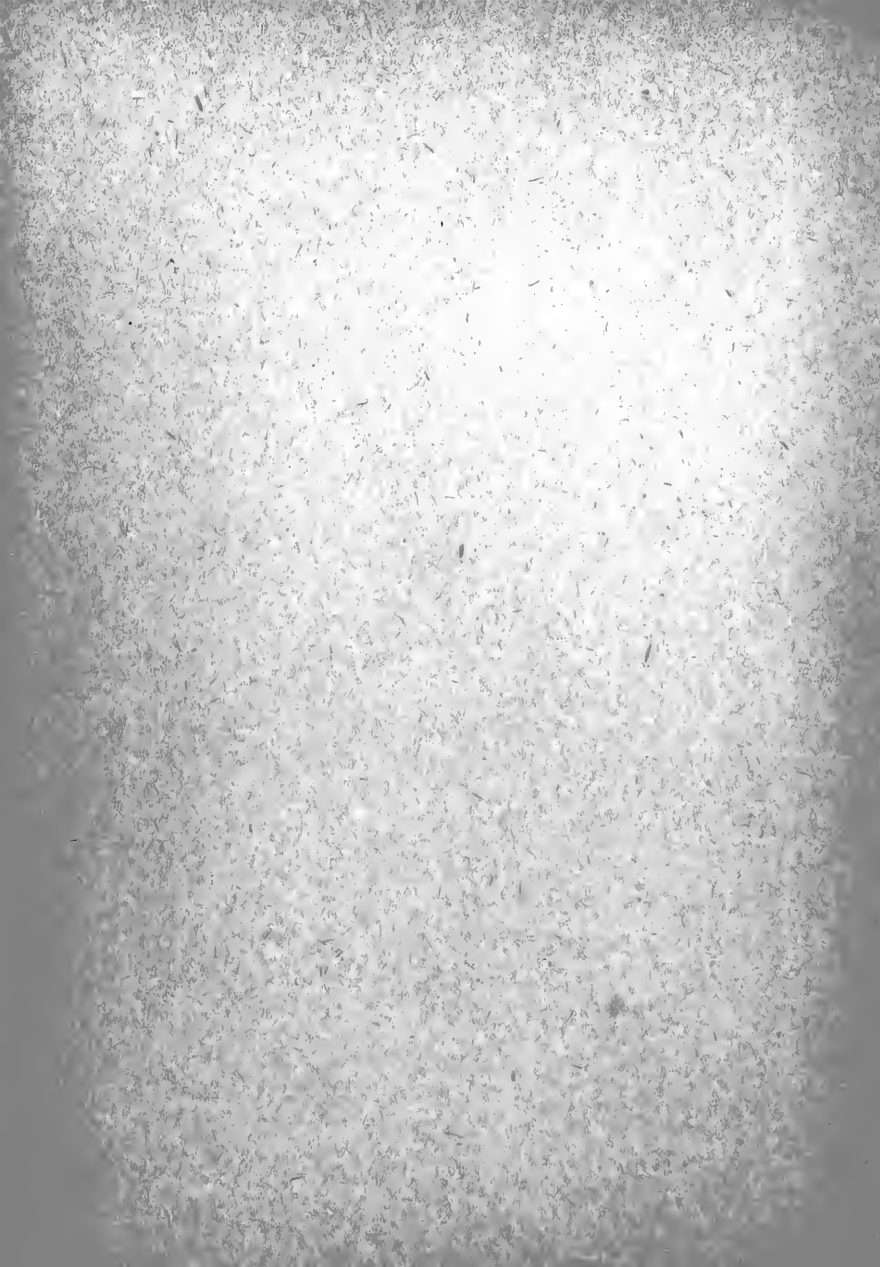
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CHOLERA,

ITS PROTEAN ASPECTS AND ITS
MANAGEMENT.

BY

DR. G. ARCHIE STOCKWELL, F.Z.S.

(Member New Sydenham Society, London.)

IN TWO VOLUMES—VOL. I.

"Respice, aspice, prospice."



1893.

GEORGE S. DAVIS,
DETROIT, MICH.

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1893.

DEDICATION.

To Dr. Cyrus M. Stockwell, of Port Huron, Michigan—the kindest and best of fathers; the most thorough, exemplary, and painstaking of tutors; the companion of youth and manhood; who early inculcated the necessity of close habits of observation and ratiocination, including self-segregation in all matters scientific—this volume is dedicated in deepest sympathy and love by

THE AUTHOR.

December 1st, 1892

PREFACE.

When this volume was undertaken, on the spur of the moment and at the solicitation of the publisher, it was not expected to be more than an exponent of personal views; but in seeking collateral evidence, I was agreeably surprised to find myself by no means singular in an estimate of cholera—that many had recognized in greater or less degree the rôle played by the nervous system, and even admitted the possibility of profound toxicity of the cerebro-spinal centres. Hence was necessitated a work more extended in scope, and more generally particularized in outline.

Again, I am pleased with the opportunity of contributing my *mite* toward undoing the evil wrought by the greatest medical heresy of any age—a heresy that seeks to elevate to the acme of pathological knowledge, a vain, visionary, theatrical egoist, devoid of even the shadow of medical training. The exponents of bacillar pathology depend solely upon hypothetical assumptions, ignoring all forms of evidence not adduced by themselves. With them the microscope is no longer an accessory to skilled observation, but may supersede the latter altogether. With profound contempt for biológico-zoölogical laws and their applications, factitious maladies, artificially produced, are made to replace real maladies. Their pathology is merely an experimental experience admitting of neither negations or offsets; their therapeutics, a form of still hunt with untried weapons, in an unknown jungle, after a hypothetical prey. Indeed, it is a sad travesty upon medical science when authors and would-be teachers wantonly assert Rabies, Cholera, Yellow-fever, Dengue, Tetanus, Endo-carditis, Pneumonia, etc., are “dis-

eases whose microbic origin is positively known;" when two of these are supported only by manifest fraud, in two more the evidence has never been adduced in any form, and in the other three it is of the most flimsy, superficial character.—The list might be considerably increased as regards the latter. The tendency is to sacrifice truth to temporary self-aggrandizement; to assert individual preferences as established facts, regardless of results, forgetting Science is a stern mistress who permits neither preferences or personalities, and refuses to acknowledge evidence that is not freed from the errors of coincidence and ably supported by negatives.

In conclusion, I will here make special acknowledgment of indebtedness to Doctor Alexander Harkin of Belfast, Ireland, for copies of his researches into the ætiology, pathology and treatment of cholera, which lead anything hitherto published upon this subject; his views are so in accord with my own, that frequently the former have by preference been adopted *in toto*, consequently the chief merit of this work will remain with him—not me.

I have also availed myself freely of the labors of Felix von Niemeyer, Thomas Hawkes Tanner, and Hermann Lebert, whose cholera essays are models of diction and profound epitomes of information concisely expressed; also of the researches of Surgeon-Majors T. R. Lewis, D. D. Cunningham, J. M. Cunningham, E. A. Parkes, and J. C. Hall; Sir Wm. Aitken, Sir Thos. Watson, Geo. Johnson, M. J. von Pettenkofer, G. V. Black, A. M. Brown, Geo. Crookshanks, Rudolf von Jaksch, James Gagney, *et al.*

I will likewise acknowledge special courtesies at the hands of Dr. Thos. C. Minor, of Cincinnati, and the Secretary of the Tennessee State Board of Health, whereby was obtained a large portion of the material embodied in the Appendices.

VII

Thus the volume makes little pretense to originality further than as a condensed rescript from others, supplemented by personal views as evolved by practical observation and experience.

GEO. ARCHIE STOCKWELL.

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CHAPTER I.

HISTORY.

In the ranks of the medical profession, no less than in lay circles, the subject of Cholera* is one of supreme and absorbing interest, especially now that, in epidemic form, it is knocking vigorously at our very gates, so to speak, and sedulously endeavoring to force an entrance.

For a number of years Americans have solaced themselves with the idea that modern science had effectually barricaded cholera from Europe, and consequently from the Western Hemisphere, except in the sporadic form that is always more or less prevalent in Mediterranean ports, and occasionally asserts itself in the United States as a concomitant of the heated term. But, while the general progress of sanitation in the civilized world has been considerable, and such as in great measure to remove the faulty conditions under which the disease flourishes, scientists are rudely awakened by the fact the danger line has by no means been eradicated, that the barriers of protection have been silently and unexpectedly forced, and that if immunity of the American continent is to be secured it is only at the price of "eternal vigilance" coupled with more perfect knowledge.

* From *χολᾶς*, "the bowels," and *ῥέω*, "to flow"—not, as has been imagined, from *χολή*, "bile," and *ῥέω*.—S.

When are recalled the fearful and weird tales of various visitations, and the universal panic that in each instance has resulted, it is little wonder the public await breathlessly each successive cholera bulletin, fearing the dread malady has broken bounds and, consequently, may at any moment manifest itself in the very midst of the most remote community.

Evidently the disease has prevailed for untold centuries in portions of the Orient, and manifested itself as both an endemic and epidemic in the Indian Peninsulas, where—until quite recently at least—it has ever been regarded as a filth disease. It is only within the last three-fourths of a century, however, that it has manifested any predilection for Europeans, prior to this period being universally regarded as a plague peculiar exclusively to dirty, badly-housed, ill-fed "heathen." In Siam and Burma cholera is always present; in Hindustan it is wont to ravage certain districts with great regularity, being in the main an accessory to religious pilgrimages, shrine-worship, etc., especially when any considerable number of natives are assembled together. It remains a regular visitant at the annual festivals held at the great Conjeveram Pagoda, forty-five miles from Madras, whence the latter city is regularly infected, and at the great gatherings of Allahabad. It was a constant attendant upon "The Jagannath," as held at Puri, a town of 35,000 inhabitants, that was generally healthy in June and July, but invariably developed cholera the succeeding

month with the influx of some 150,000 pilgrims, who filled the lodging-houses almost to the point of suffocation, and crowded even the streets and fields, literally covering both with their urine and excrement the decaying odors of which terribly impregnated the atmosphere for miles about; here the disease claimed its victims, not by scores but by hundreds, yet speedily ceased its ravages on the dispersion of the multitude and subsequent thorough amalgamation of their filth with the soil.

It was only in the early part of the present century that the malady first appeared in Jessur, in Bengal (sixty-seven miles north-east of Calcutta), as an epidemic, infectious, pestilential disorder, and overstepping its natural boundaries began a deadly march northward and westward that did not cease for seventeen years, and is supposed to have claimed nearly 600,000 victims. Sir Archibald Allison thus tells the tale :*

“After the signature of the treaty of alliance with Scindia, November 5th, 1817, the cholera, then for the first time known in British history, broke out with the utmost violence in Lord Hastings’ army, and from the very outset committed the most dreadful ravages. The year had been one of great scarcity; the food crops were of inferior quality; the situation of the British cantonment low and unhealthy. Every-

* “History of Europe,” vol. vi. Edinburgh, 1865.

thing was thus prepared for the ravages of the epidemic, which soon set in with terrible severity. For ten days the camp was nothing but a hospital; in one week 764 soldiers and 8,000 camp-followers perished. At length the troops were removed to higher and more airy cantonments, and upon this the malady ceased—a memorable fact for the instruction of future times. As was afterwards experienced, the ravages of the pestilence were greatest among the lower classes of the people. Only 148 Europeans perished in November, but about 10,000 natives fell victims to the malady during the same period. When it spread to Calcutta it destroyed 200 a day for a long time, chiefly amongst the worst-fed and most destitute people.”

So far as can be gathered, the epidemic that now threatens this country, and is already afflicting portions of Western Europe, in March or April of the present year appeared in the northern portion of hither India, near the head waters of the Ganges. It first manifested itself in a region a hundred miles or so from Mirut, and two hundred and fifty north from Lucknow, among the pilgrims at the great Hurdwar Fair. Thence it moved northward by Lahore into Kashmir, reaching Peshawar, showing itself violently at Srinagar and in the Punjab in May, a month later ravaging Askabad after traversing the whole breadth of Afghanistan. Here, having reached the Trans-Caspian railway, it moved more rapidly, securing two

points of distribution—one at Baku via the Trans-Caucasian railway, whence it reached Tiflis, Persia, on June 26th; the other at Astrakhan, at the mouth of the Volga, where it broke out June 30th, and was carried by steamboat traffic up that river as far as Tzaritzyn, Saratov, Samara, Kazan, into the Province of Kostroma, even spreading to Perm and Nijni-Novgorod, and to Ekaterinburg in the Oural. Moskva (Moscow) became infected August 5th, St. Petersburg twelve days later, when the German ports quickly caught the epidemic, which probably entered both by crossing the Prussian frontier (rail route) and by sea (via the Baltic).*

Since its inception in the Northwest Province of India, not only has it traveled the course just indicated, but it likewise covered or overflowed many thousand square miles of territory, disseminating itself in very much the same way as did the terrible epidemic of sixty years ago; and all the epidemics that have afflicted Europe, both before and since, appear to have closely adhered to the same line of progression, showing that epidemic history tends to repeat itself.†

This is a matter worthy of more than passing at-

* It is to be remarked also, that Hamburg has ever appeared a dangerous point of distribution for Western Europe.—S.

† It is notable, both the present epidemic and that of 1867, began at the Hurdwar Fair, which is said to attract as high as three millions of pilgrims each season.—S.

tention, since the present plague, unless abruptly stayed in its course, may possibly (though it is not probable) in its ravages duplicate those of 1829-34. It may be added, moreover, that the malady follows precisely the same laws in Europe and America as in the Orient; that it adheres closely to trade routes, and advances from the various points where the seed is sown (for reasons hereinafter explained) with intervening intervals of uncertain duration, in proportion to the facilities of communication; that it is in no inconsiderable degree dependent upon, and influenced by, seasonal and atmospheric conditions, dying out when these latter are modified or removed. Further, since it is only too plainly apparent that land quarantines and sanitary cordons, which European nations are ever ready to enforce against their neighbors, have not been successful in keeping out the epidemic, the utterance of Prof. S. L. Pisani, who is one of the most practical as well as sanguine sanitarians, possesses double weight: "Experience has taught that it is not in our power to prevent the importation of the germs of cholera, and that on good sanitation we should exclusively rely; let the soil be sterilized; let the seed fall on barren rock, and it will not germinate whether the season be favorable or not."

According to Dr. Wm. Farr,[†] and also Dr. J. C. Morton, cholera has probably always existed in spor-

[†] "Report on the Mortality of Cholera in 1848-49." London, 1852.

adic form in England and Europe, and this fact only serves to add fuel to the flame of an epidemic. They cite several "plagues" as presenting choleraic characteristics, among others that so carefully described by Sydenham in the seventeenth century, but which, however, appears to have more nearly approached a dysenteric flux.*

*See Appendix A.

CHAPTER II.

EPIDEMIOLOGY.

The medical world is to-day as ignorant of the primary derivation of cholera as ever, though numerous and varied hypotheses are not wanting. It is possible, perhaps even probable, that in its natural home, in the great river deltas of lower Bengal and of Siam and Burma, where it is endemic, it results from *miasm*; but whether or not this miasm is developed on diseased rice, as has been suggested, it must be admitted the disease, in its more malignant form at least, was originally exotic to Europe and America, and even now depends for its vigor, more or less, upon the constitutional dyscrasiæ of individuals, and for its persistence upon certain telluric, meteorologic, atmospheric, and concomitant unsanitary conditions.

It is especially noticeable, all others things being equal, that epidemic cholera flourishes best—that is, attacks more people, is more fatal, and more rapidly extends itself geographically—under two special conditions:

First.—During a high temperature of air and earth:

Second.—At periods when the variations of ground water and temperature are capricious, abrupt, without warning.

Thus, in October, and early November perhaps, in the northern temperate zone, when the majority of people are day by day vacillating between light clothing and heavy wraps—when colds, influenza, and malaria, are especially rampant, and other diseases prevalent that are the outcome of carelessness, deficient body-temperature, or overheating,—the ravages of cholera are apt to be greater than in December, when heavy clothing and heated houses are the rule. So too, the epidemic is more active in July and August, when the days are insufferably hot and the nights cool with heavy dews, than in either September or June. Again, the history of the disease in Asia, Europe, and America, evidences that in high and dry situations, in cold climates, wherever there is a moderately uniform or gradually progressing or regressing temperature during the twenty-four hours, its spread in the main is limited and slow; while in low-lying moist places, especially in hot climates that present extremes of humidity between meridian and the succeeding sunrise, its ravages are apt to be most severe and oftentimes quite uncontrollable. In both farther and hither India, it has been observed the pestilence suddenly springs into activity with the arrival of the southeast monsoon which, charged with humidity from the Pacific Ocean, frequently causes a fall in temperature of as much as fifteen to thirty degrees within almost as many minutes.

Further evidence of the part played by telluric,

meteorologic—and likewise astronomic—conditions, is had in the fact that when cholera is endemic to volcanic regions, it becomes more active and virulent in those seasons when there is an accession of volcanic action, or when such is pending; the disease was exceedingly active and pernicious throughout the Indian Archipelago and the Malay Peninsula after the Sumbwa eruption, and all enteric maladies of choleraic nature were then greatly increased and obtained new impetus. In Middle and Eastern Europe, when the epidemic of 1830-34 was at its height, it was observed, birds, quadrupeds, fish, and even insects, suffered, both prior and during the epidemic, from unknown maladies that caused their death in great numbers; the same was true in India; also of the United States, in varying degree according to topographical features and surroundings. Dr. O. D. Norton, of Cincinnati, recalls that in that city, in 1849, when cholera was especially virulent, birds died in their nests, and even the house flies and mosquitoes were exterminated. The same phenomena have accrued to certain portions of Europe the present year. Again, each successive cholera epidemic that swept the civilized world, has been preceded or accompanied by profound disturbances within the solar system, this year it being the occultation of Mars; also by evidences of famine: Finally, the great epidemics of influenza of this century, though their appearance may be coincidences merely, are

certainly suggestive in that in each instance, viz., in 1805-34; 1847-49; 1851-53; 1864-65; 1890-91, they preceded an epidemic of cholera. Sir Thomas Watson especially notes this fact; and John McLean for twenty-five years a Hudson Bay factor, observed the same in the peninsula of Labrador, where the natives (Nascopies and Innuits) were first decimated by influenza, then ravaged with an epidemic of choleraic character; further, it is somewhat striking that both maladies, to use the words of Watson, "issuing from their cradle in the East, traversed the northern countries of Europe till, arriving at its western boundary, they divided into great two branches; the one proceeding onward across the Atlantic, the other turning in a retrograde direction toward the south and east." Between the two epidemics moreover, there is marked similitude or analogy, the main differences being that, whereas one spared but very few and was seldom fatal, the other smote very few, but with so deadly a stroke that the death rate was exceptionally high. Both are in a sense general epidemics, affecting the whole system but especially manifest in the nervous portion; in both the most prominent symptoms are referable, in the majority of cases, to the mucous membranes—to those of the air passages in influenza, to those of the alimentary canal in cholera.

Surgeon-Majors T. R. Lewis and D. D. Cunningham, who investigated cholera in India for eleven years consecutively (1869 to 1880) under orders from

Her Majesty's Secretaries of State for War and India, remark,* that in manifesting a marked partiality for a soil of the character of the Brahmaputric and Gangetic alluvium, "cholera is by no means singular, for it is a well established fact that malarious fevers and kindred disorders flourish with most vigor about the deltas of large rivers all over the world," . . . but they would not, however, "be understood to imply that the causes productive of malarial fevers and cholera are identical, or that the localities providing the conditions necessary for the development of the one must necessarily provide those for the other also."

Speaking of the Andamans, where this malady has never flourished and malaria is always rampant, Dr. Lewis says: "Notwithstanding the Islands are within three days of India, and twenty-four to thirty-six hours of Burma, and that during the last twenty years steamers have constantly passed between the two countries and the Settlement, . . . it is only on rare occasions that cases of cholera have been registered as occurring."

Of these rare cases Dr. Rean, principal medical officer of the Settlement, says "the patients were generally admitted from some feverish locality, or had been employed on works of an unhealthy character."

The importance of well authenticated cases of this nature can scarcely be overrated in connection

*"Physiological and Pathological Researches." London, 1888.

with the ætiology of cholera; they strongly evidence the correctness of the views promulgated by both Chas. Macnamara* and Max von Pettenkofer,† and assimilate, so to speak, the two. Questions of possible infection or of water-contamination by specific imported material, can hardly be seriously entertained here. With the restrictions surrounding this isolated convict settlement there can be no casual importation of cases, as the recent history of every person is accurately known. Similar seizures, moreover, occur habitually in every city of India, as well as every summer and autumn in all the large cities of Europe, and also in America, but excite no special comment unless an epidemic supervenes, or is already rampant, when these otherwise ignored cases are seized upon, collated, and described as foci of the pestilence. It is not the custom *then* to regard such cases as due to a localized generation of the disease, and the fact the *comma bacillus* may be detected is held conclusive evidence, ignoring the well established fact that this microbe is by no means pathognomonic, but present even in conditions of health.‡

* "A Treatise on Asiatic Cholera," London. 1870.

† "Die Verbreitungsart der Cholera in Indien, nebst Atlas." Braunschwig, 1871.

‡ At the moment of this writing it is announced cholera has found foot-hold in the city of New York, the evidence resting solely upon the presence of the *comma bacillus*. That this is, presumptively, an error is shown by the press reports,

That in the present stage of knowledge it is impossible to explain all the phenomena of cholera distribution by telluric or meteorologic conditions, may be allowed, yet neither can such influences be denied. It must be remembered the same difficulties obtain in regard to malaria and kindred diseases, and that one has as substantial claim to this theory of diffusion as the other. And that cholera in its ætiological relations does present marked parallelism to other diseases that are dependent chiefly upon topographical surroundings for propagation, is proved by the fact even malaria sometimes breaks loose from its endemic haunts and shows itself in places where it before was totally unknown. Thus, says M. von Hertz* it “sweeps over considerable regions of country as an epidemic, and over vast sections of the globe as a pandemic. . . . It does not seem probable that currents of air are capable of carrying the poison generated to a distance of any considerable number of miles; I believe rather it is in a majority of cases generated upon the spot. It is a still more difficult matter to account for those isolated areas of malarial

that declare two patients were ill with the malady *eight days*. In exotic cholera, fatality supervenes, or convalescence begins, usually, ere the fifth day succeeding seizure has opened. Presumably the cases were all indigenous in character.—S.

* “Cyclopædia of Practical Medicine;” von Ziemssen. New York. 1874.

poison that are often confined to single streets, to one side of a street, or even single houses.”*

It is a matter of common experience that removal from a locality in which cholera exists is a remedy against the spread of the disease, and the East Indian Government has for many years acted on this knowledge, with regard to its troops and convicts, with gratifying success. It is equally a matter of experience the disease is most virulent in those years when, owing to telluric and meteorologic conditions, the food crops are more or less a failure and famine threatens; and that, while it manifests itself with unusual severity

* That the parallel between malaria and cholera is much more close than generally imagined is, however, evidenced in that both depend for their phenomena (as is shown of cholera in Chap. IV, p. 56, and Chap. VIII, pp. 105, 110) upon disturbances of the vaso-motor system.

The contracted vessels of the skin and the rigors associated with the cold stage in malaria, are evidences of hypertrophy and hyperæsthesia of vaso-motor nerves; while increased temperature, flushed surface, full pulse, and the dilated blood vessels accompanying pyrexia, exhibit tissue paralysis, both nervous and muscular. The splenic and hepatic engorgements, and the diarrhœas and dysenteries that are so frequent sequels of malarial poisoning, are derived from dilated and paralyzed arteries, and consequently excessive flow of blood to undilated, enfeebled tissue. In cholera, too, we have paralyzed blood vessels; but there is also another dangerous factor, in a measure specific and dependent upon the former, in that the blood itself is constantly and rapidly being deprived of its serum.—S.

in certain localities, in others closely contiguous its ravages are comparatively mild or wholly absent.— How often has it been observed in the case of an outbreak that shifting a ship a few hundred yards from its anchorage, or crossing to the other side of a river, has sufficed to end an epidemic !

An instance in fact may be cited in the visitation of America in 1853-54, when Sarnia, Ontario, and St. Clair, Michigan, suffered severely, while Port Huron, just across the river from the former and twelve miles above the latter, had but three cases, and these, there is every reason to believe, obtained the infection in Canada.

At this period neither of these towns was provided with sewers or any form of drainage other than afforded by natural topography, and the fermenting and decaying "sawdust pavements" of the streets of Port Huron, it might be supposed, would naturally tend to foster the epidemic. But the real reason for the immunity was, doubtless, the dwellings for the most part were confined to a sandy or loamy porous soil overlying a substratum of blue clay, the latter with a dip of from twenty to thirty feet to the mile, sloping toward the St. Clair River. Sarnia and St. Clair both rest on an outcropping of clay that was baked and seamed by the hot sun of a summer supervening upon a wet spring.

Again, on November 9th, 1817, cholera attacked the camp of the East India Company troops stationed

on the borders of Scindia; this cantonment was on the *right* bank of the Sindh or Betwa river; but the ravages were stayed as by magic when the forces were moved over to the *left* bank, a distance of not more than three-eighths of a mile.

It is also interesting, in this connection, to know that in India, in 1819, the citadel of Jaragurth, situated in a slight depression 1,000 feet above the level of the plain, lost many of its inhabitants, while a city near the foot of the mountain, with good natural drainage, entirely escaped attack !

Another peculiarity more or less positive in its evidence is, that while certain districts are exempt during any one epidemic, or any series of epidemics, the same may on a subsequent occasion be attacked, though there is always a decided predilection for some localities at all times. It is not uncommon to find the epidemic passing over large tracts of country, with the wind perhaps in its very teeth, and it seldom spreads itself on any systematic or geographical plan, since it may appear simultaneously in regions a thousand miles apart.

Indeed, nothing can be more capricious than the variation in the intensity of the malady in different places and at different times, or at different times in the same places. An imported case may end in a local attack confined to a single room or single house; even a simultaneous importation of a number of cases at different points may exhaust itself in a number of

local (circumscribed) epidemics; while at other times a single case suffices simply to produce a general epidemic or even a raging pestilence. The history of different epidemics in large cities shows the greatest variety of effect, according as the cholera poison found the conditions for development more or less suitable.

And when the disease is fairly established as an epidemic, its spread in a severely infected place is by no means general, or in anyway uniform. A row of houses, a series of streets or blocks, or perhaps a ward or other section, becomes an epidemic centre. Then, again, there are individual room (or several room), epidemics, sometimes with a certain preference for damp cellar lodgings; or individual groups of houses are attacked in one street; often only one side of a thoroughfare is ravaged, or out of a series of blocks perhaps only one complete square and one or two streets will be visited, while all about in the vicinity there will only be, here and there an isolated case, or none at all. Here is illustrated the combined effect of importation and of local fixation of cholera germs in the ground or drinking water, in the moisture of the walls, in the damp, heavy, musty air of unventilated rooms, and in the emanations of sewers; while the dissemination is effected by adhesion of the germs to the washing, bedding, vessels, etc.

What the primary factor may be, then, is unknown, but the fact remains that the production and

course of the malady are so greatly under the control of sanitation that neglect of measures essential to the latter, on the part of individuals, and municipal, county, and state authorities, as well as the general government, is little (if any), less than criminal.

Even without knowledge of essential cause, we are perfectly familiar with results and effects, and these afford the text upon which to work. We do know, aside from telluric or meteorologic conditions,* that

*The arguments for and against the contagious nature of cholera are many and varied, and some of the positive are the result of misunderstanding and misapprehension. Thus certain German writers are frequently quoted as contagionists, when, in fact, more careful perusal of their writings show they are only infectionists; this error arises from the fact there is but one term (*Ansteckung*) in the German language to express both conditions.

Surgeon-Lieut. Colonel J. M. Cunningham, Health Commissioner of India, who for years has studied cholera where it is endemic, emphatically declares it is not contagious. Dr. Edward Goodeve, who likewise has had extended experience in various portions of the Orient, insists the malady "does not spread from the sick to the well by any rapidly acting emanation. Surgeon-General Chas. Hunter, in his report upon the epidemic in Egypt in 1884, is equally assured of its non-contagious nature. Many others have noted that patients ill with the disease may be attended, washed, lifted, etc., with very little risk, and that the discharges from stomach and bowels are the chief, if not the only, sources of danger. Hermann Lebert pointedly remarks (Ziemssen's "Cyclopædia, of the Practice of Medicine," vol. i):

the disease, though not in strict sense contagious, during epidemic times is, in considerable degree, at least, infectious, the poison apparently being constant in the dejections of cholera patients; that this poison may be disseminated at points remote from the ravages of the malady by being carried thither in the intestines of individuals, who perhaps present no evidences of cholera other than a slight intestinal flux, hence have no idea they are victims, or being made involuntary means of communication; a transient traveler may thus, through a single privy or water-closet, infect a whole community.

Niemeyer tells us that in 1848 a detachment

“Cholera can be spread only by contagion, that is by germs which are carried from a diseased to a healthy person; but these germs infect only comparatively rarely by intercourse or contact with cholera patients, since they possess relatively but little vitality in the air of the sick-room, and are present mostly in inconsiderable quantity. On the other hand, a certain number of the germs and a given vitality are necessary for the propagation of the disease, and these conditions are better met in fluids than in the air; hence contagion is more frequent when the germs are communicated through a fluid than when transmitted through the air.”

Thus it seems, while Lebert apparently pronounces for *contagion*, he really means what in English would be *infection*. Felix von Niemeyer (“Text Book of Practical Medicine,” vol. ii.) expresses himself in like manner, and subsequently adds he is a non-contagionist, later explaining his position, making him in fact an *infectionist*.—S.

of recruits from Stettin, where cholera was raging, came to Magdeburg, two of whom on the night of their arrival fell ill of the malady, and were immediately sent to the military hospital without coming in contact with the inhabitants. Nevertheless, a few days later cholera asserted itself first, in the house where they had sojourned a few brief hours, and later along the street on which the dwelling was situated—all from the use of a privy by one of the unfortunates. Again he remarks:* “A small epidemic in Greifswald gave me excellent opportunity for observing the spread of cholera, and in almost every case I could find that the patients had used the privy of affected houses, or that they had used a privy in common with persons from these houses who had diarrhœa.”

F. D. Alexandré tells of a soldier attacked with diarrhœa, who arrived at the village of Haime, from Paris where cholera was raging, April 4th, 1849, and remained three days at his father's house, when he went to hospital—there was no supposition of cholera in his case, so light was the attack: In the course of ten days seven persons in the household contracted the malady, four of whom died. Fred'k Wm. Goëring corroborates with an account of a vagabond, suffering in like manner, who was committed to the workhouse at Dieburg, with the result the epidemic

*“Text-Book of Practical Medicine,” vol. ii. New York, 1884.

swept through that institution, but nowhere else manifested itself in the town, save in a single instance; the exception was the woman who acted as laundress to the prison.

Also, Prof. von Pettenkofer relates the case of a man committed to the prison of Ebrach, from Munich, during the existence of cholera at the Bavarian capital, and who suffered from intestinal flux. His diarrhœa persisted, though its nature was not recognized, and he was sent to prison hospital. Immediately the epidemic developed within the institution, the first victim being a female prisoner who had washed the clothing soiled with the diarrhœal discharges of the Munich convict.

Of the pernicious effects of general or common latrines may be cited the fact that, during epidemics in America, the soldiers of the U. S. Army and of the Marine Corps suffered more severely than any other relative number of people; the closets or privies in connection with barracks, are usually in a foul state, and in most instances are merely open trenches with but a rude shelter to protect from the weather.

Since it has been shown that the malady is only transferred to healthy persons—persons possessing no special or abnormal receptivity—through the dejections of those afflicted with the disease, the previously enigmatical and apparently contradictory observations concerning the spread of an epidemic are satisfactorily explained. Further, that it spreads more rapidly than

formerly is accounted for by the fact the facilities for travel and communication are greatly increased; and it is little wonder the generally traveled routes exhibit the greatest ravages, or that extension is against wind, or by long leaps with occasional retrocessions, while places intervening escape—traveling cholera-victims infect only those places where they leave their dejections. Again, if the cholera germs were contained only in the dejections of those who suffer from the severest form of the disease, as they cannot travel, long springs of cholera epidemics could only occur through spontaneous generation, or when persons infected with the poison traveled during the period of incubation, and the disease in them did not assert itself *en route*. But besides such, numerous examples prove that persons suffering from simple choleraic diarrhœa (as in the Magdeburg cases cited by Niemeyer) and who at no time are very ill, carry with them the fatal germs, and by infecting a single water-closet or out-house may start an epidemic *de novo*, as it were.

Lebert believes even a healthy person may carry the germ from locality to locality, and yet suffer no inconvenience—*i.e.*, he may escape the malady altogether. He calls attention to the fact one seizure also renders individuals in some degree personally immune, though their aptitude for carrying the infection is in no way lost providing they have been exposed to its influence. While he declares his doubt whether

the cause is an organic poison or living organism, he is inclined to accept the mycetic theory which, as he remarks, "explains without strained effort why it is that fluids, and especially stagnant fluids, containing more or less organic nutritious matter, are chief vehicles of the cholera germs, as they are of all proto-mycetic forms. It is on this account that the water of the soil, the drinking water, and every fluid, play so highly important a rôle in the diffusion of the disease; and yet neither the ground water or the drinking water theories can ever prevail in sole sovereignty as causes of the disease, since such are not necessary for the development of the germs, but only become so when they can furnish these germs with proper nutritious matter, when other favorable conditions of growth are presented, and when more especially the way of communication with the human organism is open. The germs of cholera may be spread without ground or drinking water just as easily as with them, through the air, by becoming attached to solid bodies, etc. . . . True, cholera finds in drinking water also a very frequent and most potent medium of dissemination, as it may be impregnated with the poison (from water of the soil by filtration from privies and sewers) which may then flourish in further development; still drinking water alone cannot be considered as the exclusive or necessary means of dissemination. Over-flowing or badly cemented drainage or sewer pipes, for instance, conveying infectious matter, may

carry their foul contents directly into the ground walls of cellars, and dwellings, and swiftly develop destruction among the inhabitants."

It is probable, however, the dejections do not when first expelled contain the cholera germs in the stage of development necessary to infection, but that they become strictly aggressive only after having undergone fermentation, which result is furthered by admixture with decomposing animal substances—and this is why the midden privy is always a greater source of danger than the modern closet. This theory is supported by numerous facts.

Dr. O. D. Norton, a veteran practitioner of Cincinnati, who had extended experience with cholera in the epidemic of 1849, remarks, regarding experiments with *fresh* alvine excretions of "rice-water" character, that he and a confrère fed such by *buckets-full* to numerous chickens and pigs, but induced in them no evidences of the disease; on the contrary, these creatures seemed to "grow fatter" thereon.*

The observations of C. von. Thiersch show that while recent dejections are not dangerous to animals, feeding the same with *old* excreta of the same sort invariably induces the malady.†

Further, experience shows that physicians passing

* Cincinnati Lancet-Clinic, vol. xxiv; Sept. 24th, 1892.

† "Meine Cholera-Infektionsversuche vom Jahre, 1854, und die des Herrn Dr. B. J. Stokvis vom Jahre, 1866." Munich, 1867.

from bedside to bedside are comparatively immune.— Niemeyer says his experience in cholera epidemics, wherein he wrapped patients in blankets and often held them in his arms for some time, made him a “decided anti-contagionist;” that those who wash the body and bed linen after they have lain some time, are more apt to be infected than those who directly care for the patient, even to removing the dejecta.

Again, Lebert says: “I have noticed in all epidemics, and have seen it mentioned in the writings of many authors, that practicing physicians, even hospital physicians, are seldom attacked with cholera.”

In Cairo, Egypt, in 1831, of one hundred servants employed as rubbers or *masseurs* of cholera patients, not one was ever attacked; of eighty rubbers at the hospital at Mansurah, and sixty at Damietta, all escaped save one. None of the physicians or nurses of the cholera service at Constantinople in 1855–56, and Oran in 1861, ever suffered from any form of the disease.

Washerwomen, whenever they wash linen soiled with cholera dejections, without any precautions, are attacked in all places in no small numbers.* In Branson,

* The frequency with which washerwomen fall ill with the disease from contact with infected linen has often been mentioned, but there are also examples where cholera has been spread by rags and other objects. The same is true in still higher degree of unclean bedding. C. von Zehnder ascribes the origin of two cholera centres in the Zürich epi-

in the Canton of Valais (Switzerland), in 1867, one of the Sisters of Charity nursed, with the greatest self-sacrifice, all the cholera patients in very filthy chambers, and yet remained healthy, but at the close of the epidemic "her sympathy prompted her to assist in washing up the soiled linen, when she was attacked with the disease and died. It was from a washer-woman, who died after washing the clothes of a cholera fugitive, that the epidemic developed later in Zürich, 1867." (Lebert.)

Very numerous facts might be cited to demonstrate that cholera may be communicated, and carried from place to place, by clothing or other material soiled by cholera dejections; the observations of Etienne Moulin, Gaston Pellissier, Jas. Simpson, Jules Bucquoy, J. M. and D. D. Cunningham, Max von Pettenkofer, Antoine Fauvel, Augusto Guastella, and others, are most definite. Guastella remarks:† "There were persons living in places sheltered from the epidemic who, after washing linen soiled with the dejections of cholera, carried the disease afar." Fauvel adds other facts‡ showing that camping places where an

demic of 1867 to an accumulation of bedding, mattresses, pillows, etc., that had been used on the beds of cholera patients, and afterwards piled up, before being carried off for disinfection, in the neighborhood of the houses affected.

—S.

† "D'igiene e medicina navale ad uso della marina mercantile." Trieste, 1861.

‡ "Le Choléra; etiologie et prophylaxie." Paris, 1868.

epidemic has occurred, hospital wards, sick chambers, ships and cars carrying cholera patients, etc., may preserve for some time, under certain circumstances, the power of transmitting the disease; nevertheless, such examples are comparatively rare. To transmit cholera by clothing, he considers, demands certain conditions, viz.: "To transport it a short distance requires certain contact with objects in connection with the patients, especially those soiled by vomit and rectal discharges; to transport long distances, the objects previously exposed to contact must be confined to close quarters where the fresh air is not renewed, and where sunlight does not enter. There are few examples of objects freely ventilated carrying the disease for any long time, or long distance, while there are many cases to prove that the transmission may easily occur where soiled effects have been closely packed for several months."

As to the influence of dead bodies in disseminating infection directly—*i. e.*, by handling,—Lebert expresses himself as doubting it very much. "We occupied ourselves almost daily in Paris, in 1849—my friends and myself—with investigations into the pathological anatomy of cholera. In Zürich, in 1855, I made all the post mortem examinations, with my assistant, Dr. Wegelin, and neither of us, and no one of our dead-room attendants, were attacked with the disease. I consider it, therefore, merely an accident when a body-carrier falls sick. I believe, indeed,

that animal putrefaction rather diminishes the capacity for infection, and that the bacteria of decomposition destroy the germs of cholera."

Hugo Wilhelm von Ziemssen* lays especial stress upon the fact "it is more dangerous for the persons in a house if the evacuations are emptied into a privy filled with excrement, into a cess-pool, or thrown on a dunghill, as in such places the germs seem to find circumstances most favorable to their development and increase."

A. von Hirsch† insists marshy and malarial regions are especially favorable to the dissemination of cholera, in that they furnish nourishment for the germs, favoring their multiplication; also because the soil in such localities is eminently fitted to transmit, by soaking and slow filtering, cess-pool fluids and sewage waste, carrying into cellars and basements; that thus the privy of a neighbor may be more dangerous than one's own, especially if in close proximity to the residence of the latter.

From observations made during the last three epidemics in France, Dr. Hippolyte Mireur concludes cholera is not transmitted directly from the ill to the well by contact or through the respiratory passages; that the products emanating from cholera patients—

* "Die Choleraepidemie, vom Jahre 1867." Greifswald, 1870.

† "Rückblick auf die neuere Choleralitteratur." Schmidt's Jahrbücher. Bd. lxxxviii.

the dejections and vomited matters—alone contain the germs, which are not immediately transmitted by themselves, but when placed under favoring conditions give rise to an infectious principle; that clothing and merchandise,—such as skins, hides, rags, etc.—much more than individuals, are the agents for the transportation of this principle. *

If then there is any justice in the belief of Lebert, Hirsch, *et al.*, that the cholera germ lies within a spore,† it is more than probable the ripening of such in the fæces after evacuation is the real source of infection; or that the product of the ripened spore, on being returned to an economy, further develops producing perhaps certain alkaloids that, in turn, taken up by the absorbents, induce violent toxic symptoms—symptoms that, made manifest through the nervous system, constitute the phenomena of the disease. Dr. Thos. King Chambers remarks:‡ “There is every reason to believe the chief exciting cause of the disease is a poison generated by decomposing

*“Etude historique et pratique sur la prophylaxie et le traitement du choléra,” etc. Paris, 1884.

† The question of a spore is an interesting one from a certain standpoint since the majority of observers of and believers in, deny such to the comma bacillus; yet Huppe, of Prague, declares this germ does possess “a fructification propensity by virtue of an arthrosporulation,” which he personally observed.—S.

‡ “The Renewal of Life; Lectures Chiefly Clinical.” London, 1864.

organic matter and received into the body from without. To judge by its effects, it seems widely diffused through the air, especially in the neighborhood of its origin—in the air of privies, cess-pools, sewers, putrid marshes, and crowded human habitations. One is perhaps tempted to ask how it is, if the poison is spread so broadcast, that everybody does not get poisoned; but it must be remembered two things are necessary to poisoning, viz.: Not only a poison, but a person in condition to be poisoned; and in point of fact *the latter* is the more important element in the transaction."

Thus the weight of evidence goes to show cholera epidemics, for existence and dissemination, demand three prime factors, viz.:

First. Conditions of soil, atmosphere, etc. (general surroundings), favorable to the nourishment of the germ or germs, which would otherwise speedily lose the power of infecting:

Second. Conditions in each human subject, individually, favoring receptivity:

Third. Direct infection *per se*:

And regarding the last, it is believed the principal, if not the only way of insuring infection, is through the medium of the intestinal canal and its absorbents:

That the germs are ordinarily carried but a short distance through or by the air:

That the great danger lies with the alvine evacu-

tions and vomited matters, but only after fermentation has been set up therein:

That contamination of the water supply and of food, by cholera discharges, is ever a grave factor:

And, finally, that linen, cotton, or woollen fabrics, soiled by cholera discharges, if excluded from air and sunlight, serve to keep alive the germs of the disease for an indefinite period.—The history of the barque *Swanton*, on which cholera did not appear until she had been at sea for twenty-seven days, when clothing was unpacked by the passengers, also of the ship *New York*, on which the disease did not manifest itself until she was sixteen days out from her port of departure, and then under the same circumstances as on board the *Swanton*, both evidence the truth of this statement.

CHAPTER III.

TRANSMISSION DANGERS.

It is to Prof. von Pettenkofer we are indebted for the discovery that porosity of the soil, by enabling the contents of privies and cess-pools containing the cholera germs to freely permeate and soak the ground for some distance around, and poison wells and sewers, favors the rapid extension of the disease, while the opposite quality to some extent inhibits dissemination; and the same author was the first to demonstrate that the "manifest fitness of any locality for the disease depends on excrement, containing the germs, permeating the soil and exposed to circumstances favorable to decomposition."

Next to soaking of the soil is the danger from gutters and drains, which may carry the infection from house to house; and it is well known that a soil-pipe, or untrapped rain-gutter, has carried the disease into an uninfected dwelling through a window of the latter being contiguous to, and at higher elevation than, the upper end of the latter.

There can be no doubt foul drinking water plays no inconsiderable rôle in dissemination. Mr. J. Snow* established the connection of the fearful local epi-

* "Cholera and the Water Supply in the South Districts of London in 1854." London, 1856.

demic in Broad street, St. James' Parish, London, in 1854 with an infected well; its ravages ceased when this supply of water was shut off from the public. J. Simon* declares in the portion of London supplied with river water drawn from the stream after it had received the contents of a large number of sewers, so that it had forty-six grains of solid constituents to the gallon, the number who succumbed to the malady was thirteen out of every thousand, while in other situations, under precisely parallel circumstances and surroundings, save the water supply contained but thirteen grains of solids to the gallon, the death rate was only 3.7 per 1,000. Edward Frankland,† speaking of the same city and relation of water supply to cholera, says:

“On the 18th of August, 1866, a family removed from London to Margate; on the 26th there was a storm with heavy fall of rain, and the water had an unusual odor and taste. On the 27th four persons were attacked with cholera, and on the following day still more, the most of whom died. The water in the well at the end of the garden furnished, in 100,000 parts, 93.4 of solid matters, of which 7.36 parts were of organic or volatile nature. The cess-pool adjoining the garden had clearly poured its contents into

*“Report on the Two Last Cholera Epidemics as Affected by the Consumption of Impure Water.” London, 1856.

†“The Water Supply of London and the Cholera.” Quarterly Journal of Science, 1867.

the well after the overflow caused by the rain, and this had caused the fatal contamination, for an analysis made Sept. 18th showed 82.75 solids (in 100,000), of which but 1.13 parts were of organic or volatile nature. It was proven that all who were attacked had drank from the well. A similar occurrence was established by Dr. Lancaster, of Epping Forest."

The same author declares the inhabitants of London who used Thames water from Kew, above the city, showed a mortality from cholera of but 8 in 10,000; those who used the water from Hammer-smith, 17 in 10,000; from Battersea to Waterloo Bridge—that is water contaminated by the sewage of the city—163 in 10,000. In 1854 only the half of a district was supplied from Teddington Loch, and the mortality therein was 87 in 10,000, but in 1866, all the water in the Loch having been drawn off, the mortality was less than one-tenth of that in 1854. Again, in 1866 the cholera was very severe in the East End, which was supplied by the East London Water Company, from Oldford, the reservoir, on the river Lee, being little better than an open excrement and sewer receptacle, even filtration being neglected. The result was the mortality in this portion of the city was from 63 to 112 per 10,000, while the balance of the London, with a pure water supply, exhibited a death rate of only 2 to 12 per 10,000.

Manchester suffered terribly from cholera in 1832 and 1849, when the water supply was very impure,

but in 1854 and 1866, the water being derived from the interior of Derbyshire through an aqueduct, there were very few cases, and these only of a sporadic character.

Dr. W. Schiefferdecker, too, mentions a fact worthy of note concerning the six great cholera epidemics that ravaged Königsberg, Prussia, from 1831 to 1866, in which more than 2500 people succumbed out of nearly six thousand attacked.* The inhabitants of those portions of the city supplied with drinking water from the river Pregel, and from wells, were those that suffered most, while those supplied by a system of water-works from the so-called "upper tank," in which the water was exceedingly pure, suffered much less severely; the Pregel and wells were fed with ground water and sewage.

Dr. J. Grätzer also describes an instance occurring in Breslau during the epidemic of 1867,† in which the walls of a badly constructed privy attached to a newly built and well arranged house rendered the water in an adjacent well impure; besides, the privy vault was not regularly emptied, and its contents overflowed into an unwalled excavation in the neighborhood of a large accumulation of ground-water. The consequence of this contamination, which affected the drinking water was, that in the beginning of the

*"Die Choleraepidemie vom Jahre 1871 in Königsberg." Königsberg 1873.

†"Die Breslauer Cholera-Epidemic." Breslau 1873.

epidemic no less than twelve of the inhabitants of the house were attacked, *eleven* of whom died; also other persons in the vicinity, who obtained water from the same well, were seized. In this instance, it was proved the cholera poison first entered the privy, thence passed into the ground water, then into the drinking water, and so on into the digestive organs of the unfortunates.

Again, when cholera is once introduced it sometimes happens only those are attacked who are in the house where the first infection is received, or who visit the same closet; and in some instances the malady has been restricted solely to house epidemics—further illustration of the care that should be exercised in the way of sanitation.

Niemeyer believes the poison is rarely taken into the system in the drinking water, but in the main enters the economy during the act of respiration and, lodging in nose, mouth, or throat, is swallowed with the saliva. "Using infected privies is so dangerous, because they are the favorite lurking-places of cholera germs, and the gases arising always contain dust-like particles." The poison passes from the closet to the dwelling, and A. Biermer insists the latter are "more liable than individuals to infect."

So far I have said nothing, relatively, regarding the claims of two schools the members of one of which, like sheep, blindly follow their bell-wether over any obstruction regardless of cause or reason. First

of these stand the followers of Louis Pasteur—a visionary whose utterances have never been worthy of dependence, who is utterly devoid of physiological or other medical knowledge, and who, moreover, is not even a reputable chemist—the bacillary pathologists. Second, those walking in the footsteps of Francesco Selmi and Armand Gautier, the latter of whom stands first in the discovery and investigation of vital alkaloids, and has had courage to approach some problems of physiology and pathology most abstruse and complex in nature, opening up a wide territory that has hitherto escaped investigation. It might be added there is a third coterie, who, between the “devil and the deep sea,” have appeared anxious to reconcile the foregoing, and by blending the two evolve a new pathology, Emanuel Klein, perhaps, being the most able representative of the class.

Practical medicine has suffered much from the invasion of new theories as well as experimental methods; traditional pathology has given way to the experimental, and spontaneous maladies to those forms that may be artificially provoked in inferior creatures. Further, the upholders of bacteriological pathology have followed too closely in the steps of their masters, in that they sedulously ignore all negative evidence while magnifying the positive. For instance, the monumental report on cholera just issued from the Government press at Washington, was com-

piled by an ardent bacteriologist, who, while gathering everything favorable to his view, carefully excluded all evidence calculated to invalidate; he quotes the early reports of Surgeon-Major D. D. Cunningham, which are indefinite, and sedulously ignores the later investigations of the same author as well as those of Surgeon-General Wm. R. Cornish, Surgeon-Major Timothy R. Lewis, Professor Ray Linkster, Sir Wm. Aitken, Doctor A. N. Brown, *et al.* —an act which renders the “sin of omission” even greater than that of commission. *Ætiology*, theoretically, has been very much simplified by the discovery of microbes, but certainly scientific medicine has very little profited thereby, since attention has been diverted from clinical observation and research. The fallacious charms of the germ theory have caused medical men to forget their mission, have prevented the relief of the ill, and produced misconceptions of disease. Dr. B. W. Richardson* only recently protested against the idea it was necessary “to subject a patient to a kind of modified snake-bite in order to settle a question of diagnosis,” and adds: “Twenty years ago the profession was steering well and steadily towards great principles on the preventive, as well as curative side of medicine; then crept in the wild enthusiasm of bacteriological research — research good enough in its way as a piece of natural history,

*The Asclepiad, 1891.

and as disclosing some curious tribal phenomena developed under morbid states of the organic structures and the blood, but a positive insanity when accepted as the one absorbing pursuit, restoring humoral pathology, ignoring nerve function, leading to Babel with its utter confusion of tongues, and separating for a time the modern art of cure from the accumulated treasures of knowledge, wisdom, and light of over two thousand years."

It may here be remarked, likewise, that the majority of maladies to which popular pathology ascribes bacterial origin, are very severe in character, and most generally fatal. Therefore, in cases where dissolution is rapidly impending, it is not without the bounds of probability or possibility—indeed has been triumphantly demonstrated in many instances,—that partial death may affect the fluids and tissues to greater or less extent preceding the general death of the organism; that is to say the sum of the deaths of the constituent elements. Again, in certain cases, changes usually observed *post-mortem* may and do take place in the fluids and tissues, or a portion thereof, *ante-mortem*—sloughing, gangrene, phthisis, malignant diseases, etc., are evidences of this. Consequently, bacteria, which under natural surroundings and habits of life are ever the products of decomposition, may be found in the tissues, blood, and other fluids during life, and be the results merely of the advanced degree of the diseased condition—not the

cause of its development. And to force inductions as the result of artificial cultivation of microbes, and the artificial diseases induced thereby, instead of through natural development of the same and the pathological factors under which such development occurs, is not alone contrary to all scientific precedent, but must always remain a source of error. Just as tame fruits differ from the wild, and domestic creatures from those that are *feræ natura*, so must bacilli differ according to their mode of cultivation and development, and in their results; a parallel under such circumstances cannot but be fallacious, as every zoölogist or botanist well understands, and a more thorough knowledge of these branches of science, on the part of the medical profession, would lead to fewer errors in pathological investigation.

Again, Dr. Burdon-Sanderson's investigations* prove the development of infective inflammatory products as the result, purely, of chemical irritants, while Lewis and Cunningham alike observed bacteria in the blood of creatures dying from such experiments. These important facts have a most pertinent bearing on the ætiology of cholera, as will be observed in a subsequent chapter.

In the report of the *German Cholera Commission* dated Calcutta, February 2d, 1884, Koch announced the comma bacillus as the specific cause of

*The Lancet (London), vol. i, 1873.

cholera; and since his claimed discovery was based on microscopic slides from four specimens of cholera in natives, said slides being furnished by Surgeon-Major T. R. Lewis, the latter, in conjunction with Surgeon-Major D. D. Cunningham, deemed the matter worthy of further investigation. In their report it is remarked:*

“So far, therefore, the selection of the comma-shaped bacillus as the *materies morbi* of cholera appears to be entirely arbitrary. Dr. Koch and his colleagues have adduced no evidence to show that it is more pernicious than any other microbe; indeed, as a matter of fact, the sole argument of any weight which has been brought forward . . . is the circumstance it is more or less prevalent in every case of the disease, and that the *German Cholera Commission* had not succeeded in finding it in any other.”

With regard to the suggestion that the cholera process may in some way favor the growth of these bacilli, and that they are not necessarily a cause of the disease, Dr. Koch, in the report from Calcutta, declares such a view is “untenable,” inasmuch as it would have to be assumed “that the alimentary canal of a person stricken with cholera must have already contained these bacteria; and, seeing that they have been invariably found in the comparatively large number of cases of the disease in Egypt and India, it

*“Physiological and Pathological Researches.” London, 1888.

would be necessary to assume further, that every individual must harbor them in his system. This, however, cannot be the case, because, as already stated, the comma-like bacilli are never found except in cases of cholera."

To this Drs. Lewis and D. D. Cunningham reply,* if Koch and his colleagues had submitted the secretions of the mouth and fauces—the very commencement of the alimentary canal—to careful microscopic examination of the same kind as that to which they have submitted the alvine discharges, "we feel persuaded that such a sentence would not have been written, seeing that comma-shaped bacilli, identical in size, in form, and in reaction to anilin dyes, with those found in cholera dejecta, are ordinarily present in the mouth of perfectly healthy persons."

Koch subsequently admits he had examined the mouth of healthy persons but found no comma bacilli; on another occasion, as claimed by C. S. Dalley† (though I can nowhere find such utterance in Koch's own publications), he declared his familiarity with the comma bacillus of the mouth, and that it differs from his cholera bacillus in "being longer, more slender, and not so blunt at the ends," etc.; but T. R. Lewis, Douglas Cunningham, Ray Linkster, Arthur E. Brown, and Sir Wm. Aitken subsequently proved the identity

* *Ibid.*

† "Technology of Bacteriological Investigation." Boston, 1885.

of the organisms by accurate tests and measurements applied respectively to colonies taken:

(a) From the mouth of healthy human beings ranging from four to fifty years of age:

(b) From the alvine discharges of cholera-affected persons:

(c) From the intestines of persons who had died of cholera:

(d) From cultivations of all three in agar-agar jelly, in weakly alkaline peptone gelatin, etc., and:

(e) By the reaction of all to staining fluids—fuchsin, gentian violet, methylen blue, etc.

Further, during a subsequent and independent investigation, D. D. Cunningham found ten different kinds of comma bacilli in the dejections of sixteen consecutive cholera patients, that of Koch "being very far from the most numerous of the lot." Lewis, too, found the Koch bacillus most abundantly in the drinking waters of India in the season when the people using such waters were absolutely free from cholera. And M. Neller* observed the bacillus of the present cholera epidemic in Europe to differ markedly from that claimed by Koch to have been discovered in India, in that it is thicker, shorter, and larger than the latter, causes turbidity of bouillon, and in peptonized gelatin grows more rapidly; it was found in twenty-nine cases of cholera, also in the

* Le Progrès Médicale. 1892.

sputum of a case of broncho-pneumonia; and in thirty additional cases of cholera, *no comma bacillus whatever* could be found.

The result of these investigations conclusively prove the so-called cholera-bacillus to be only an "old friend under a new name"—an everyday *spirillum*.

Again, the investigations of Pettenkofer,* Biermer, and others, are founded on clinical data too

* Prof. von Pettenkofer who, together with Robert Koch, has been actively engaged in studying and observing cholera in the present Hamburg epidemic, declares that the latter's theory of the origin of the malady "has not stood the test of experience." It has not been proved that the pestilence was brought to the city in the way indicated by Koch, or that the comma bacillus is the cause of the epidemic. Commenting editorially upon this, *The Lancet* (London) remarks: "One of the difficulties attending the acceptance of the cholera bacillus theory of the causation of cholera is, to account for the occurrence of isolated and sporadic cases of that disease, which in the aggregate amount to a large number—in India, for example. One man is attacked and dies of cholera in the barrack room occupied by a number of others, the air, food, water supply and all other conditions being the same for all alike. There is perhaps no other case of cholera in the station at the time, nor is there any history of any having occurred before; and this is not at all an uncommon but a frequent occurrence in India at certain seasons, and outside and beyond the so-called endemic area. The relation of these cases to larger outbreaks and epidemics has not hitherto been exactly defined, for when the epidemic occurs later on, it does not at all follow that it should be at the

absolute for disapproval, while Koch declares:† “In these experiments, as to the influence of development-inhibiting materials, the surprising fact was established that comma bacilli extraordinarily *easily die when they are dried*,” and again—“For the spread of the infective material the main condition is, that the dejections should remain in a moist state, for *as soon as they dry up they lose their activity*.”‡ And laboratory experiments and experiences have demonstrated these bacilli are among the most sensitive and non-resistant of organisms of their class, since a temperature of either 15° or 50° C. (59° or 122° Farh.) is sure death thereto within a *very brief* period of time; moreover their resistance to chemical agents is almost *nil*, especially in relation to acids, hence their destruction would be assured in the stomach by means of the gastric juice if the latter is of normal reaction; only in an alkaline or neutral medium is it possible to secure development. Koch tacitly admits this, and Klein and Herman Bigg, and the whole host of followers are compelled to corroborate. Further, the

same place or time or soon after the occurrence to which we are referred; and parallel with this inquiry, how did the River Seine become infested with the comma bacillus, and how was the cause of the disease introduced into Hamburg, or, to take the latest example, into the well at Portel, the fishing village near Boulogne?”—S.

† Report of July, 1884—“Cholera in Europe and India,” by E. O. Shakespeare, Washington, 1892.

‡ Italics mine.—S.

fact may be recalled that while cold in the main seems to inhibit certain epidemics of cholera—as those of 1849 and 1866—in certain other instances it appeared to have no effect; in 1830 and 1831, in the height of a severe Russian winter, its ravages were of a most virulent nature, in Moscow with a temperature of *minus* 4° Farh., and even in Orenburg with a temperature of *minus* 22° Farh.

If, then, the comma bacillus is the cause, infection should be much more active at the bedside than in the closet—among physicians and nurses than laundresses, scavengers, and those who handle and cleanse the dried and soiled bed-clothing and body-linen,—whereas the precise contrary has been repeatedly proven by the best of all tests, practical experience. (Thiersch, Niemeyer, Tanner, Flint, Watson, Lebert, *et al.*) Again, it is a well known fact cholera dejections may be boiled to the utter destruction of the last bacillus, yet be not deprived of their virulence; (Virchow, Aitken, Lewis.)

Arnaldo Cantani and Klebs repeatedly obtained cholera poisoning from sterilized cholera fluids, and Lewis and D. D. Cunningham as far back as 1874 made a like observation. Finally, Jno. Simon, A. Delpech, Wm. Sedgwick, Dutrieux Bey, Alex. Harkin, J. M. Cunningham, Lionel Beal, Thos. J. Mays, B. W. Richardson, John Chaine, Mariano Semmola—the latter recognized as one of the most profound pathologists of our century—and others, attach no

importance to microbes, which they believe at most to be only modified anatomical elements, and sequels of the pathology instead of a cause thereof; that they are accidental accompaniments of disease, of which nothing can be predicated of the action of any particular form; they are scavengers only.

Thus is confirmed the trite utterance of Trichum, *à propos* of Koch's bacillus, when he adjured his listeners not to believe the purported discovery settled the question of cholera any more than knowledge of the tubercular bacillus would eradicate pulmonary phthisis. Semmola adds:* "This doctrine at best is based only on a hypothetical basis;" and, "I cannot comprehend how true clinicians can accept as of practical value results that are established solely in the laboratory."

Again, the Koch theory of infection embodies the belief that when a patient fails to succumb under an attack of cholera, it is owing to his organism having not only withstood the onslaught of the bacilli or the virulence of their morbid products, but that the invading hordes must have perished within the body. But that these hordes are a sequel rather than a cause, has just been shown by Dr. Cornet, of Berlin and Reichenhall, who discovered that persons apparently convalescent from the malady may carry about with them in their intestines, active living Koch bacilli.†

* Berliner Klinische Wochenschrift. 1891.

† "The case in which this important discovery was

Regarding the teachings of the Gautier school, it may be said they have little in common with pan-germic and bacterial theorizings, but rest upon data positive, precise, easy of verification; and if the indications submitted meet with the attention they seem to deserve, a sweeping reformation will result.

Both Gautier and M. Peter, following the lead of the great Selmi, believe there are elements resident within the economy that may induce certain specific diseases; that these elements remain inoffensive whilst elimination and oxydation of detritus is normally operative, but give rise to disease if from some cause or other this elimination and oxydation are interfered with whereby the detritus accumulates and exerts a toxic influence upon the nerve centres; in fact, that not only after death, but even during life, the animal organism—in accordance with physiological and chemical processes readily determined—has the power of elaborating a numerous class of alkaloids essen-

noticed was that of a man whose mother, wife, and son died of cholera. He himself had a slight attack, and was put under the care of Dr. Carl Lauenstein, of the Seemann Hospital. He was nine days in the hospital, . . . recovered perfectly, and was on full diet. Last Friday he was up and anxious to go home, but was induced to stay. On Saturday he was still better, and no motion at all, and it was with difficulty he was induced to remain. On Sunday Dr. Cornet discovered that in the stools passed there were large quantities of comma bacilli."—*British Medical Journal*, Oct. 8th, 1892.

tially toxic in properties, those evolved from dead tissue being termed *ptomaines*, those from living tissue *leucomaines*.* But this is not all. Gautier has also ascertained that in the living animal economy there are elaborated azotized uncrystallizable salts, substances the precise character of which is still undetermined, and which are the extractive matters (toalbumins?); and while the ptomaines and leucomaines are both highly poisonous, the extractives are far more toxic than either! This discovery of ptomaines, leucomaines, etc., though perhaps of comparatively little value from a therapeutic standpoint, is nevertheless of the greatest importance to pathology.

The importance of such authoritative teachings has not escaped the germ theorists, who are forced to admit the symptoms that supervene upon a cholera attack obviously indicate a form of poison. Even Koch, along with others, has been compelled to hesi-

* Ptomaine, from *Πρῶμα* a "carcase" or dead body, and *ivo*, "material"—or *in* from Latin, *inus* "belonging to." Leucomaine from *Λεύκωμα*, anything whitened as albumen or white of egg.

This nomenclature is far from satisfactory. The selection of the term *ptomaine*, indicative merely of the conditions under which animal alkaloids were first discovered, as a root whence to derive a name for these bodies, is too restrictive, since it is only appropriate for alkaloids of post-mortem origin. A title is still needed for alkaloids formed by morbid processes during life—the *ptomaines of disease*.—S.

tate, and to inquire if some ptomaine is not a specific cause, even while claiming a microbic origin for the disease; and Klein strongly supports this view. Dr. A. M. Brown,* who is vouched for by Gautier as an English exponent of his ideas, remarks:

“Cholera, . . . monopolizing as it does so much scientific interest, supplies the finest field for airing the respective claims of the two pathological theories, . . . the one bacillar, organic, and strictly biological; the other toxic, inorganic, and strictly bio-chemical. The first, so high in favor, and with the entire field to itself, has failed egregiously in its assumed solution. The various expeditions undertaken—Indian, African, and European—in bacterio-bacillar interests, have proved as practically hopeless and unprofitable as the discovery of a north-east passage to Cathay. By such missions Koch has only added to his hypothetical perplexities, while Klein and Bouchard, with modified appreciation, preserve their germ proclivities, and hope by vested but truly humoralistic concessions to solve the cholera problem.”

Koch thought he had found the pathogermic entity, but, confronted and constrained by cold *facts*, has later felt himself obliged to modify his positive utterances, and consequently now admits the comma bacillus does not directly engender cholera, and that it

* “Animal Alkaloids—The Ptomaines and Leucomaines.” London, 1889.

can only do so indirectly by the intervention of a ptomaine which he supposes it secretes. Thus he seeks to ally himself with the third class, who endeavor to reconcile theory with fact by preaching the "Good Lord, good Devil" doctrine! And this must imply two suppositions—first, a specific bacillus which Koch *has not* discovered; second, the secretion of a ptomaine by that bacillus which the Berlin savant and his following are equally as far from discovering.— Says Sir Wm. Aitken,* *à propos* of the foregoing:

"A. G. Pouchet obtained an oily base belonging to the pyridin series from cholera stools, and Brieger got from pure cultivations of the comma bacillus in beef broth, in addition to the common ptomaine of putrefaction, two poisons which he regarded as specific products of this bacillus." But none of the poisons which have been thus isolated produce exactly the symptoms of cholera: In Cantani's experiments, tremor, prostration, spasms, and repeated vomiting were observed; Klebs noticed muscular contractions and alterations of the kidney; the poison obtained by Pouchet irritated the stomach and slowed the heart; one of L. Brieger's produced muscular tremor and cramps, while the other induced lethargy and feebleness of circulation with occasional bloody diarrhœa. These facts, remarks Aitken, evidence "the symptoms of cholera are not caused by a poison formed by the

* "Animal Alkaloids." London, 1889.

action of the comma bacillus, and it is evident that much more extended investigation is required before the pathology of the disease is accurately understood." C. H. Fagge* suggests that in all such investigations one must bear in mind the possibility of the poison being formed, *not* in the intestines merely, but in the blood, nerves, and general tissues.

—Thus Koch assumes, Gautier demonstrates, and Klein and Chas. Bouchard† vacillate while sedulously essaying to promote fusion.

* "Principles and Practice of Medicine." London.

† Since the above was written, attention has been called to an editorial in the Boston Medical and Surgical Journal for July 28th, 1892, from the pen of my friend Dr. E. P. Hurd, of Newburyport, Mass., as follows:

"The toxic theory of Bouchard, as set forth in his book 'On the Auto-Intoxications,' fairly well explains the symptomatology of cholera. He demonstrated, as early as 1884, by experiments made with toxic substances found in the stools and urine of cholera patients, that the pathogeny of cholera may be referred to multiple intoxications.

"Professor Bouchard has some doubts as to the fact of the comma bacillus being the pathogenic agent of cholera. 'The only serious argument,' he says, 'in favor of Koch's claim, is the presence in the intestines of cholera patients of special micro-organisms, which are not supposed to be found in the intestines of healthy persons or of persons affected with other diseases. These micro-organisms exist often in considerable abundance, from the very first, and often to the exclusion of every other microbe in the digestive tube. Apart from this empirical ascertainment, which warrants only a presumption, all the other arguments which have been alleged are illusory.'

"The toxic alkaloids which Bouchard has extracted from the intestines and urine of cholera patients, greatly ex-

ceed those ordinarily contained in fæcal matters. One of these, which forms acicular crystals, seems to have a special virulence, and to be identical with the 'cholera poison' which Koch and Brieger have isolated from the intestinal contents of cholera patients, and which they believe to be generated by the comma bacillus. There is, however, no agreement as to what really are the soluble toxic substances secreted by the microbe of cholera. Bouchard affirms that the real virus is eliminated in the urine in appreciable quantities. In injecting into the veins of animals cholera urine, he has caused pronounced cyanosis, collapse, albuminuria, anuria, cramps, and pale, yellowish or bloody diarrhœic evacuations, like those which characterize true cholera. In injecting the alcoholic extract of the urine of cholera patients, he has determined somnolence, albuminuria, diarrhœa, and death in two days.

"There is," says Bouchard, 'in cholera-urine a poison which I call the true cholera poison. I cannot chemically define it; I only know by its physiological properties. I know not if it is fabricated by the sick organism or by microbes.'

"Bouchard's view then is, that besides the primary infection there exists in the pathogeny of cholera a secondary intoxication, consequent on the infection. He thinks that the symptoms considered as characteristic of cholera are the result of this intoxication. To this we may attribute the cyanosis, the chilliness, the respiratory troubles, the hic-cough, the special diarrhœa, the intestinal desquamation, the cramps, the de-hydration of the blood and tissues, the albuminuria, the anuria. But very soon 'there supervenes a new source of systemic intoxication superadded to the first, and this clinically expresses itself by intellectual torpor — by somnolence, apathy, and coma. The respiratory rhythm changes, sometimes rising, sometimes falling; it is the rhythm of uræmia. The pupils are contracted, and become punctiform.'

"This is evidently a different symptom-aggregate from that of the initial period, and is due to another kind of poisoning; in other words, we have the clinical tableau of uræmia from excess of disassimilation and blocking of the kidneys.

"In short, cholera furnishes us an example of a double auto-intoxication; one by an abnormal product,—this the

choleric intoxication properly so called; the other by normal products,—constituting a variety of uræmic poisoning.' ”

From the foregoing it would seem Bouchard is gradually “ undergoing a change of heart.”—S.

CHAPTER IV.

PATHOLOGICAL DISCUSSION.

Setting theories aside, I may now deal with real facts. With all the wrangling of pan-germists, biochemical physiologists, and fusionists, one thing is most evident, viz. : The neurotic character of cholera !

As far back as the time of Wm. Cullen, who was upheld by Sir Thomas Watson, the disease found place in nosological nomenclature under the "*order* Neuroses, *class* Spasms." Jules Marey always considered the nervous system as primarily affected by cholera poison, and as determining the principal phenomena of the attack, even the gastro-intestinal symptoms. The poison, he declares, first excites the sympathetic system, whence ensues the contraction of the muscles under the dependence of that system. The spasm of the arteries of the greater and lesser circulation, as well as of the bronchial radicles, explains the phenomena of the cold period. In the period of reaction, the arterioles and capillaries relax, and there is stasis of the circulation and excessive watery exudation. And everything manifestly points to profound toxication of nerve centres inducing changes that are chiefly revealed through the great sympathetic, particularly in its abdominal and thoracic area.

Sir Henry MacCormac* and Chas. Lever,† both of whom had extended experience in Ireland in the epidemic of 1834, alike regarded the malady as provoked by a lesion of the sympathetic—a view supported by Wm. Sedgwick, Jas. Johnson, Claude Bernard, D'Arsonval, the elder Chermak, Fillipo Picani, D. Cannataci, Foster, Guérin, and Alex. Harkin.

But it is evident we must look even further. Manifestly the gastric and enteric pathology is not primary, but secondary, since in “dry cholera” (*cholera sicca*; *cholera asphyxia*; *cholera siderans*) death supervenes ere there is any evidence of intestinal or stomachal disorder or distress, and through failure of the respiratory and cardiac centres. Indeed, the latter factors are so prominent in every attack that Drs. E. A. Parkes, Jonathan Hutchinson, Geo. Johnson, J. Snow, W. Grissinger, and Surgeon-Major J. C. Hall, were inclined to believe the real morbid factor exists in the blood whereby is induced spasm of arterioles inhibiting pulmonary circulation and preventing oxydation, laying especial stress on the fact that there is likewise evidence of spasmodic contraction of the circular organic muscular fibres of the bronchi. Says Dr. Parkes:‡ “That there is some

*“Observations on Spasmodic Cholera; Its Origin, Nature and Treatment,” etc. London, 1834,

†“Cholera in the South and West of Ireland.” Dublin, 1834.

‡“Researches into the Pathology and Treatment of Asiatic or Algid Cholera.” London, 1847.

impediment or arrest of the circulation in the capillary system generally, and in the pulmonary capillaries in particular, appears almost certain; and it is by no means improbable, from the whole bearing of the facts, that this is due to chemical change in the fibrin and in the mode of its combination consequent on the direct action of the active cause."

Evidently the latter "builded better than he knew," and had a partial insight into the truth, as is seen in the terminal portion of the above sentence.

Dr. George Johnson, as cited by Thos. Hawkes Tanner, remarks: "During the state of collapse the passage of the blood through the lungs from the right to the left side of the heart is in greater or less degree impeded."* But he differs from Dr. Parkes as to the cause of this impeded circulation, his hypothesis being that the poisoned blood causes contraction of the muscular walls (instead of spasm) of the minute pulmonary arteries, the effect of which is necessarily to diminish or arrest the flow of blood through the pulmonary capillaries. Alburtus Eulenberg, Chas. Francois, J. M. French, and others, attribute cholera-algidity to cardiac adynamia provoked by nervous irritation proceeding from the intestine, a theory that derives support from the experiments of Tarchonoff and Franck who showed that irritation of the digestive tube and

* "Notes on Cholera, its Nature and Treatment," 1866
— "The Practice of Medicine." London, 1874.

mesenteric nerves may determine more or less prolonged arrest of the heart.

Though these views in a measure clash, they may all be considered as containing great germs of fact, being based upon observation in different individuals under variable conditions; moreover they are, in a measure, reconcilable when later pathological knowledge is brought to bear thereupon, and due consideration is given to the influence of the nervous system.

Though the theory of primary intestinal lesion has many advocates, who lay especial stress on two manifest symptoms, viz., dehydration of blood and tissues, and blood poisoning—that under the influence of the profuse watery discharges, provoked by such intestinal lesion, the blood and tissues became unfit for nutrition and functional work,—it must be remembered that in many cases no changes whatever are to be observed after death, either in the stomach, intestines, or elsewhere, save perhaps congestion of the pulmonary and cutaneous systems; but the left heart (as shown by Simon and others) is generally empty, while the right is distended and filled with blood. Sicluna and J. M. Bruce,* performing autopsies on the victims of the epidemic that ravaged Malta in 1887, always observed the cavities of the left heart empty and of the right filled with blood!

*“Treatment of Cholera.” Dublin Jour. Med. Science. March, 1890.

George Budd* reports concentric hypertrophy in cases of sudden death from cholera: and Jas. Jackson† especially noticed that at post-mortems of victims the hearts usually exhibited hypertrophy of the left ventricle.

Again, the elder Flint‡ lays stress upon the fact “epidemic cholera has no constant, appreciable, anatomical characters—none which appear to be commensurate with the gravity of the malady; the morbid appearances after death do not afford an adequate explanation of the symptomatic phenomena, nor do they elucidate the pathology of the disease.”

Tanner declares§ “post-mortem examinations have thrown little light . . .”; that “we naturally look first to the gastro-intestinal mucous membrane, but beyond distension of the follicles with serum, an œdematous condition of the mucous lining, patches of venous congestion, and here and there rupture of the vessels producing ecchymoses, we find nothing. The blood is altered more or less, is usually of tarry appearance and consistence, the proportion of water being much diminished, the fibrin being either reduced in quantity or affected in character, and the corpuscles increased, while the serum is rich in albumen, contains a slight excess of urea, and its salts,

* Medical Chronicle, vol. xxi.

† “Report on Cholera in France.” London, 1872.

‡ “Practice of Medicine.” Philadelphia, 1873.

§ “The Practice of Medicine.” London, 1874.

collectively, perhaps, diminished . . . the heart is often flaccid, its right side dilated, the left side contracted."

Niemeyer, barring those cases where death occurred during the stage of reaction, emphasizes the fact* "the characteristic changes consist chiefly in extensive catarrh of the intestines accompanied by detachment of the epithelium and copious transudation, and in decided thickening of the blood and excessive venous hyperæmia of the kidney" (the latter mentioned also by Tanner as an occasional but by no means constant feature).

Lebert remarks:† "The anatomical lesions of cholera are of peculiar character, but clearly more the *consequences* than the cause of the disease, hence possess no pathognomonic character whatever." And again: "The anatomical changes, the hyperæmia of the mucous membrane, the distension of the mesenteric veins with thick blood, the ecchymoses and hæmorrhagic suffusions of the mucous membrane, the swelling and great softening of the lymphatic apparatus of the small intestines, are, I am convinced, *not* the cause of 'rice-water' stools."

Rudolf von Jaksch, Jas. Cagney, Hoppe-Seyler, C. Schmid, C. Zehnder, Jas. Sterling, T. R. Lewis, F.

* Text Book of Practical Medicine," vol. ii. New York, 1882.

† "Ziemssen's Cyclopædia of the Practice of Medicine," vol. i. New York, 1874.

Delafield, *et al.*, corroborate these authors, admitting an inadequate pathology as evinced by the disease in any stage; and when is further recalled the fact there is no malady in the whole nosology that is more efficiently assisted in gaining a foothold in the economy, or the fatal tendency of which is more vigorously promoted and hastened by mental causes, we have most convincing evidence the ultimate source or cause of cholera lies deeper than in the organs that permit of general review and inspection.

Chas. Bouchard insists "from the study of the various attempts of pathologists to explain the symptomatology of cholera, it results that we must admit multiple causes. If the most powerful come under the head of intoxications, we must still make due account of the dehydration of the blood and tissues, and of the *reflexes* which take their start in the digestive tube and *affect the vaso-motors*. In favor of this latter influence, may we not refer to the alidity and collapses which sometimes follow the gastric crises of tabes, and which bear so striking a resemblance to cholera?"

CHAPTER V.

CHOLERA CHARACTERISTICS.

The characteristics of cholera, those most manifest in its so-called epidemic or malignant form, and upon which differential diagnosis chiefly rests, are:

First Stage.—A feeling of stupidity, general weakness, chilliness—more rarely a regular chill,—followed by uncontrollable watery diarrhœa devoid of color or nearly so, lacking also in odor. The first dejections are apt to be dark and pappy, but once the contents proper of the intestines are cleared out, they become of whey-like character, sometimes of pale reddish hue owing to admixture with blood, with not the slightest traces of bile pigment, and on standing usually deposit a fine granular, whitish-gray substance which contains triple phosphates, bacteria, fine shreds of algæ and blood-corpuscles, sometimes also phosphate and crystalloid salts of lime; this fluid is alkaline, being disproportionately rich in sodium chloride, and may contain some albumen, though not in great quantity. The investigations of Surgeon-Major Lewis evidence the flakes and corpuscles of “rice-water” stools do *not* consist of epithelium, nor of its *debris*, but that their formation “appears to depend upon the effusion of blood plasma;” that “the bodies found by Surgeon E. A. Parkes, moreover, correspond very closely in their microscopic and chemical characters, as well as in manifestations of vitality, to the corpus-

cles which are known to form in such fluid and are generally to greater or less degree associated with blood-cells, even when the presence of such is not suspected, especially when the disease tends toward fatal termination, when the latter have been frequently seen to replace the former altogether.”* This diarrhœa, which varies in frequency in different epidemics, may be regarded as the warning of an attack; and where it is absent, patients before the outbreak usually feel depressed, tired, and uncomfortable. These first manifestations, which are however some times totally absent, may be regarded as *stadium prodromorum*.

The duration of prodromic diarrhœa in cases of absolute cholera, as ascertained by Lebert, does not as a rule exceed three days, “but may continue five or even eight days.” He gives a table of thirty-five cases, closely observed during the Zürich epidemic of 1855, which is here reproduced:

DURATION OF PRODROMIC DIARRHŒA.	DIED.	RECOVERED.	TOTAL.
One day.....	1	7	8
One to two days.....	3	6	9
Three days.....	4	5	9
Five days	1	1	2
Six days.....	1	1	2
Eight days	1	3	4
Three weeks.....	1	..	1
	<hr/> 12	<hr/> 23	<hr/> 35

* “Pathological and Pathological Researches.” London, 1888.

Second Stage.—This stage, with which cholera not infrequently precipitately commences, constitutes the attack proper, and has been defined as algid or asphyctic—terms not altogether pertinent. There is a feeling of stupidity, general weakness, chilliness—occasionally a marked rigor,—followed by increased intestinal flux, the passages being expelled with great and sudden force, without warning; there appears to be complete loss of power over the sphincters. In addition to the “rice-water” evacuations, there is usually vomiting—which in many cases is a prominent and persistent symptom,—the expelled matters, like those from the intestine, being devoid of color and odor; this emesis may appear as a much more terrible symptom than the diarrhœa. A notable fact is, absence of pain, whereby the sufferer is enabled to endure the attack with comparative indifference up to the actual occurrence of cramps.

The cramps—in the lower extremities and abdomen, perhaps across loins, rendering the muscles as “hard as wood” or “drawing into knots,” as it were, the frequency of which varies in different epidemics, — constitute one of the most remarkable symptoms of the malady. Lebert usually observed in the second half of the attack proper, rarely earlier, and that they always assumed a tonic character in adults, while in children the tetanic form commonly obtained; occasionally, though rarely, the muscles of the face are involved. Each attack lasts but a few minutes, but

the frequent recurrence and excruciating character mark as the most distressing manifestation of the disease. Sometimes cramps persist to the very end in rapidly fatal cases, but usually they cease with the progress of the asphyxia, and in more protracted cases, in the cold period just preceding reaction. In a few instances both cramps and vomiting are observed together at the very outset of the malady, in conjunction with dizziness, headache, very great disquiet and anxiety—though, as a matter of fact, most patients exhibit a certain indifference. At the height of a very intense epidemic are sometimes seen patients who rapidly collapse with symptoms of great distress, becoming cold, cyanotic, dying after one, two three, five, six or more hours; but in such there is usually an abundance of colorless transudation into the intestine.

In the most intense or malignant development of the malady, persons may die pulseless, cold, cyanosed, etc., with no evidences of vomiting or diarrhœa, and with positively no characteristic changes to be found in the intestines or elsewhere, and no tangible cause for fatality; but these cases are so rare of late years that a large number of medical men are inclined to deny the existence of “dry cholera,” which was generally considered as proved in the earlier epidemics.

Thirst is invariably a most assertive symptom, and usually very urgent, though its degree is apt

to be in inverse proportion to the severity of the seizure; patients clutch at the attendants' hands as the glass or cup is held to them, in terror lest it should be taken away too soon.*

The circulation is greatly diminished, the pulse frequent and proportionately weakened, ranging from 120 to 140 per minute, though under some circumstances it becomes more and more feeble without acceleration, and in more pronounced or asphyctic conditions may fall below normal. When the state of collapse is fully developed the pulse is extinct at the wrist; next the pulsation in the carotids disappears; finally there is feebleness or absence of the apex beat of the heart, and of all cardiac sounds, evidencing greatly diminished power of the central organ of circulation. There is also general venous stasis giving rise to remarkable lividity or blueness at the roots of

* Says a correspondent of the British Medical Journal, a volunteer nurse of the Eppendorf hospital, writing from Hamburg recently:

“There are two pretty yellow-haired sisters who lie in a cot, with whom the characteristic ravenous thirst is the most pronounced symptom. They have no vomiting and but little diarrhoea. They sleep about twenty-three hours out of the twenty-four, and in their lethargy thirst seems to be the only consciousness. One lies grasping a cup with both her small hands, and if an attempt be made to take it from her, she automatically and drowsily opens her mouth for a draught, not knowing when the milk is given her, but swallowing mechanically.”

the nails, in the lips, face, and on the surface of the body generally; icy coldness of the skin everywhere— of the nose, tongue, and even the breath; frequently noises in the ears or head; dizziness, dimness of vision, deafness. The skin becomes shrivelled, and if picked up in a fold remains puckered for a time, retracting but slowly. Finally the entire surface of the body is bedewed with death-like dampness.

The number of respirations is usually increased, often to twenty-four, thirty, or even forty per minute, at the same time short, confined, and imperfect, frequently of sighing or irregular rhythm; the expired atmosphere, when collapse is complete, besides being of low temperature, contains more oxygen and less carbonic acid than in health, evidencing notable deficiency in the changes incident to the function. There is also marked alteration of the voice (*vox cholericæ*), which becomes whispering and unnatural owing to diminished volume of respiratory gases; oppression and pain at the præcordia are manifest, often of such excruciating nature as not to be accounted for solely by the dyspnœa present.—In many instances this dyspnœa is more particularly marked during the period of violent discharges from stomach and intestines and at the beginning of the absolute algid stage, only to disappear again with the conclusion of this period; pressure over the stomach usually aggravates. Cough is scarcely ever observed; and stertor is but exceptionally noticed and only in fatal

cases. In many instances complete aphonia supervenes, the motion of the lips being seen during efforts at articulation alone; and though this condition may at times yield for a brief moment, it is usually only when the intensity of the spasmodic muscular contractions causes the patient to cry out.

Nothing is more constant in this stage than the participation of the kidneys, and the manifold effects resulting therefrom. Albuminuria may sometimes supervene, the cloudiness varying from light opalescence to abundant deposit on ebullition, followed by partial or complete suppression of the renal secretion. Usually the microscope reveals a large number of wavy casts, most manifest when the urine has not been clouded by heating; also uric acid salts, as well as some blood corpuscles; and test with muriatic acid is apt to exhibit a large amount of indigo pigment (indican) which is certainly suggestive of profound disturbance of nerve centres, and also of the close relationship of the disease to malarial disorders. Lebert shows, in his report on the Zürich epidemic of 1855, that discoloration and commencing fatty degeneration may be recognized in the cortical substance of the kidneys of individuals who have died early in the asphyctic stage, and that this degenerative parenchymatous nephritis, which is more distinctly anatomical the longer it lasts, is not sufficient to explain the anuria that supervenes; further, it must be noted that the nephritis of cholera depends for its origin upon

disturbance in the nerve-centres, since it never by any accident becomes chronic, but disappears with the final vestiges of the disease. Lebert adds: "In all the four years after the epidemic in Zürich, I never was called upon to treat one of my patients for chronic nephritis, and among a great number of nephritic patients in Basel, I never found one in whom the nephritis could in any way be referred to a past attack of cholera."

It should be remarked that all the foregoing symptoms are apt to follow each other much more rapidly in children, especially under three years of age, than in adults, and death usually terminates with convulsions; all, however, may exhibit different combinations in different degrees of intensity, and so establish from the start the distinction between lighter and graver forms as well as between the numerous transitional grades. As a rule patients really suffer but a very few hours, and then, as already noted, only in consequence of cramps, since the intestinal and stomachal discharges are painless, between which profound rest occurs that closely borders on apathy. The expression of the face, in the beginning may manifest exhaustion and discomfort, but such is speedily followed by apparent indifference; later the sunken eyes, which are peculiar to grave cases, become remarkably dull and dry, and are only imperfectly covered by the lids.

The diarrhœal discharges may vary from three to twenty, but seldom exceed ten or twelve, each

amounting to perhaps four or six ounces, so that on the average the material transuded from the intestines does not exceed three or five pints; the quantity lost by emesis is perhaps much less; further, in either case the amount of fluid lost is not more copious in fatal seizures than in those which recover. The ease with which the contents of the stomach are expelled is most remarkable, since the act partakes of the character of simple regurgitation, occurring in series of efforts, repeated three, four, eight, ten, or more times. The whole period of all discharges varies between eight and twenty-four hours; they then become more and more infrequent, finally wholly cease for several hours, perhaps for a day or two, only to return, perhaps, at irregular periods; vomiting is especially apt to return after the ingestion of fluids. The absence of bile pigment in the stools seldom lasts more than twenty-four hours, when the period of reaction (third stage) sets in and they become yellowish-green; but before they wholly return to normal, there is an irregular exhibition of fæcal material varying between moderate diarrhœa and constipation, unless dysentery complicates.

There is entire absence of fever or febrile condition. The temperature is depressed, the thermometer falling to 93° and to 90° Farh.—rarely below the latter figure, though it has been known to reach 75° Farh.,—notwithstanding which the sufferer complains of oppression and prefers to lie uncovered;

generally during both collapse and reaction, the temperature in vagina or rectum (or both) is three or four degrees higher than in the axilla, which in turn is at least one or two degrees lower than in the mouth.

Malignant (asphyctic) cholera, runs a very acute course, and patients may die at any time in from two to twelve, eighteen, or twenty hours; death, however, is comparatively rare in the first twelve hours, occurring usually in the succeeding ten or sixteen, and when occurring on the second day is usually in consequence of imperfect reaction. The algid stage rarely lasts longer than two days, and the evacuations often cease some time before dissolution, though this is very far from being a favorable sign, since it is due, not to cessation of transudation, but to complete paralysis of intestinal muscles; *per contra*, patients in whom the evacuations continue for a long time, recover more frequently than those in whom they cease suddenly; consequently the occurrence of intestinal paralysis must be regarded as a most unfavorable manifestation, while, on the other hand, persistence of evacuations evidences such paralysis has not supervened, hence justifies more favorable prognosis.

Niemeyer early advanced the opinion that temperature diminishes only at the periphery of the body remaining elevated within, an assumption now generally accepted by the medical world. Through numerous careful observations of the temperature of cholera pa-

tients in the algid stage, Jüterborgk arrived at the following conclusions:

“The head, extremities, etc., are colder than in almost any other disease:

“The temperature of the cavities of the body, such as the vagina and rectum, is the highest (that can be measured) in the body, and should always be taken for measurements:

“Whether the case be favorable or fatal, the temperature within the body is usually increased, rarely normal, and more rarely diminished, although no cause for this has ever been found in the pathological symptoms during life or on autopsy—[Another evidence of the neurotic character of the disease.—S.]:

“The temperature of the whole body usually rises with the approach of death; but there are cases where this rise takes place without one being able to find any reason for this deviation:”

Again: “The commencement of reaction is not accompanied by any elevation of temperature, but the interior of the body usually cools off, while the outer parts warm up:

“In cases of protracted reaction, the temperature of the whole body generally sinks below the normal:

“The inflammatory sequelæ usually, if not always, excite decided elevation of temperature:

“During perfect convalescence, an abnormal elevation of temperature is often seen without any pathological cause therefor being discoverable.”

An abnormal and notable condition of the nervous system, is manifest from the fact that, while the intellect remains clear to the last*—and though the sufferer is sometimes quite hopeful,—there is in general an apathetic condition frequently amounting to complete callousness; there are no apprehensions, and little care as to what the ultimate result may be, though perhaps prior to the seizure there may have been intense dread of the disease.—The terrible clearness of mind and recognition of the end which is said to have characterized the earlier epidemics, has not been witnessed during the present ravages of the disease in Europe. In some cases there is great restlessness and tossing—unconscious movements to-and-fro,—though often the sufferer is quiet, and appears to experience no inconvenience save when disturbed by the evacuations, vomitings, or cramps. Delirium is generally absent, but occurs

*“ It is remarkable, notwithstanding the great debility which makes every motion difficult, and the profound prostration that is expressed in every feature,” says Lebert, “ that the patients not infrequently possess entire consciousness. This, to me, was one of the most disagreeable impressions of the Paris epidemic—to hear sufferers in whom the pulse was no longer perceptible, in whom the face was cyanosed and cold, still speaking with the most perfect possession of all the faculties of the mind.” According to Reinhardt and Leubuscher, some insane patients entirely recover sanity for the time being, though the sanity vanishes with convalescence; others remain insane to the end.—S.

more frequently among alcoholics, and later in the typhoid state (third stage) during which it alternates with sopor; in cases of pronounced uræmia, it is sometimes attended with convulsions. Strange to say, muscular strength, real or apparent, appears to persist in most extraordinary degree to the very last, and patients if not prevented will frequently get up and walk about a few moments before dissolution. Again, "walking cholera" is by no means uncommon, the sufferers keeping their feet until fairly in the throes of death, and in such cases locomotion appears to hasten fatality.

Death is usually peaceful, by asthenia and, as already noted, may take place at any time from two to twenty-four hours after the attack—it is a gradual "going out," the "rattling of the throat" which pertains to most diseases, being conspicuous only by its absence; or if surviving beyond twenty-four hours, there is usually manifest evidence of amendment. Commonly patients become lethargic, the lethargy culminating in sleep; sleep in turn is merged into coma, and coma into dissolution. As in many other severe diseases, there is usually observed an elevation of temperature as the fatal end approaches, while the exhalation of carbonic acid gas is very much diminished; the temperature continues to rise, in many instances even after death, as Davey observed as long ago as 1839—an observation that has since been confirmed in a series of other satisfactory tests. The bodies cool off very slowly.

Regarding the mortality of cholera, it may be remarked no one has ever been able to complete statistics of the graver cases, because the lighter ones for the most part escape accurate observation, while as to the more serious the mortality varies (according to the most conscientious statements), between two-fifths and three-fifths (the average may be put at one-half), though in some local epidemics under unfavorable circumstances, especially in asylums for the aged and for incurables, it reaches as high as two-thirds, or even three-fourths. Nearly one-third of the deaths occur within twenty-four hours, and about one-half of all the deaths occur in the first two days. In the neighborhood of one-sixth die on the third day in consequence of imperfect reaction, and about one-third during protracted convalescence and in the typhoid state—after from four to twelve days. In favorable cases of confirmed cholera, and in half of *all* cases that recover, convalescence occurs in three or four days; in the other half which recover, it occurs irregularly up to the second half of the first week. From the beginning of convalescence to perfect recovery, a period of from three to eight days usually intervenes, varying according to the character of the attack, and the characteristics of the individual patient and his surroundings.

CHAPTER VI.

REACTION AND CONVALESCENCE.

Third Stage.—Although in the preceding stage all symptoms may reach such intensity that a large number of those seized cannot survive, yet in other numbers a third or so-called “stage of reaction” supervenes, which exhibits most remarkable tendency to restoration of physiological function, though certainly not always with equal results; it is possible (though rarely witnessed), for recovery to be most rapid.

The first improvement is manifested by some repression of the discharges; even though emesis and diarrhœa are still persistent, the quantity is diminished and the whey-like character lost; in some instances, thus early, repression may be complete, even to a degree constituting absolute constipation. Another evidence of improvement is when the stomach fails to reject the fluids ingested, whereby assurance is had that the function of absorption is no longer in abeyance, and restoration of the fluids of the circulation, lost by transudation, possible. The capillary circulation is first renewed. Next that controlled by the carotids. Then the radial pulse (which before could not be felt or was scarcely perceptible) quickly regains its strength, and in a few hours is often stronger and fuller than in the normal condi-

tion; usually, also it is rapid, though seldom increased above ninety or one hundred, perhaps with distinct dicrotic beat. The double heart tones also soon become normal and regular, the blowing sound synchronous with the diastole, disappearing. Should venesection now be attempted, the blood will be found to flow almost as freely as in health, though of course, the proportion of serum is greatly diminished. As soon, too, as circulation is restored, the cyanosis disappears, though many patients preserve for some time a marble or cadaverous appearance. Heat gradually diffuses itself into the peripheric parts of the body, in fact often transcends a normal medium temperature; and possibly profuse perspiration may be induced, either of spontaneous character, or as the result of hot drinks; when temperature exceeds the normal, and the cheeks become suffused with dark red, the eyes also injected, lachrymose, and painful, and general evidences of fluxionary cerebral and other organic hyperæmia, a clinical picture is presented that oftentimes is most difficult to interpret; it sometimes disappears spontaneously, and again is evidence of imperfect reaction and threatening sequelæ. Such congestions are more frequent, violent, and dangerous in children; indeed, the stage of reaction is, in the main, more intense in the little folk, though of shorter duration, and demands watchful care. Even most adults complain of a feeling of cold and heaviness in and about the head, more manifest

about the occiput or sinciput; roaring of the ears or tintinabulations are common; and notwithstanding a certain tendency to somnolence, those persons most enfeebled are usually sleepless.

Cramps cease, as a rule, with the beginning of reaction; but the urine remains scanty, or altogether suppressed, for twenty-four or more hours, and always exhibits traces of albumen as soon as passed, which traces persist for from two to seven days.* Respiration is normal; dyspnœa absent or nearly so, having been markedly lessened toward the conclusion of the second stage. When the convalescence is rapid, the tongue clears off, the bad taste is lost, appetite returns, sometimes to a degree that causes error in diet and consequent relapse. The discharges from the intestines may persist, but soon assume a more

*In observations at Zürich, Lebert found the first urine after total suppression, was not passed until forty-eight hours after the beginning of the disease. As a rule it was regarded the secretion would be restored in the course of the third or in the beginning of the fourth day. "The first urine passed was, usually, small in quantity, in two cases bloody, and once attended with violent pains about the kidneys. Several hours, from eight to twelve, usually elapsed between this first and the second discharge. Specific gravity varied between 1.007 and 1.010. At first there was considerable brown coloring matter present, and on boiling with nitric acid it often showed a light bluish tint (indigo coloring matter). Only once was the first urine somewhat cloudy, without albumen; in all other cases albumen was present, and

natural color and solid consistence; the casts and albumen disappear, and progress toward health is rapid, so that the latter, barring accidents and slight after-pains, may be regarded as established in from ten to fourteen days subsequent to the primary seizure. Says Lebert, "If nearly half the patients die in the algid stage, in more than half of the rest, the stage of reaction goes on to favorable termination." In women, metrorrhagias, during or in the intervals of menstruation, are not infrequent during reaction and convalescence.

At all periods of life, especially in advanced age, reaction may be imperfect, may even be followed by a relapse to the second stage; yet many cases recover in spite of numerous vicissitudes and fluctuations: Or the diarrhoea and vomiting may recur from time to time with critical symptoms; or a dysentery may alter-

remained for three or four days and sometimes longer; the quantity of albumen varied, and when it disappeared the quantity of urine became much more copious."

According to investigations of Lehmann and Volk, confirmed by Prof Buhl, of Munich, the first urine voided is only quantitatively small and albuminous, but contains traces of sugar, a little sodium chloride, and relatively very little urea; but in the two succeeding days the quantity of urine, as well as its relative proportions of urea and salt, greatly increases, even far exceeds the normal, and then, after some variation, again returns to the natural condition, when the albumen, casts, and abnormal pigment disappear, and the specific gravity becomes normal.—S.

nate with obstinate constipation, the former green or greenish yellow and gelatinous; the tongue does not clear up; anorexia, bad taste in mouth, and thirst, continue to torture; the little nourishment taken is either speedily rejected or induces profound distress.

Catarrhal inflammation of the genito-urinary tract is especially apt to delay convalescence, and often in conjunction with, or succeeded by, a diphtheritic condition of the *prima viæ* induced by irritation of the denuded intestines or their contents. Most patients who fall into this state die of exhaustion.

Pneumonia, or so-called typhoid pneumonia, is especially apt to supervene; and Niemeyer remarks that "in old, decrepit persons, if physical examination be neglected, the outward resemblance and the subjective symptoms often cause pneumonia to be diagnosed as catarrhal fever, nervous influenza, typhus, etc." According to his experience, acute croupous nephritis, with the retention of urine it causes by plugging of the uriniferous tubules, is a common sequel of *cholera asphyxia*, but by no means the constant cause of cholera typhoid, as has often been asserted.

"If the secretion of urine remains suppressed after the disappearance of the symptoms of collapse, or if the scanty urine contains quantities of albumen and fibrinous casts for days; if vomiting recommences and the patients complain of severe headache and become comatose, or have epileptiform convulsions;

it is safe to make a diagnosis of acute croupous nephritis with so-called uræmic intoxication; in such cases the skin has occasionally been found encrusted with crystallized urea." (Niemeyer.)

If the first or second day after the cessation of the asphyctic symptoms the patients do not pass a normal or at least large amount of urine, or the albumen, at first very constant, does not disappear after a few days, they are apt to fall into a state of exceeding apathy and stupor, or muttering delirium, when the tongue becomes dry and crusted, the pulse frequent and often double, the temperature elevated, and they slip down toward the foot of the bed; indeed the condition so exactly resembles severe enteric fever as to fully warrant the title of cholera typhoid. Besides, there is usually a fœtid diarrhœa in which are discovered shreds of epithelium; and while the patients can scarcely be aroused from the comatose state, they twitch the face, or recover consciousness and complain of pain, if strong pressure is made upon the abdomen.*

If there is catarrhal or diphtheritic inflammation of the intestine, or of the genito-urinary tract, a pneumonia, a pleurisy, or other of the inflammatory sequelæ of cholera, the appearance of the patient does not materially differ from the above description. The

* So called *cholera typhoid* is one of the most common forms of protracted convalescence, and considered by Fre-
richs as a uræmic condition purely.—S.

typhoid peculiarities prevail in completeness, while the symptoms of the original or local disease become subjective, falling into the background or disappearing entirely. Finally, in many cases, neither during life nor on autopsy, is it possible to discover any local lesions to which can consistently be referred the exhausting fever, of which so many die after the cholera proper has run its course—further evidence of nerve-toxæmia.

Particular importance has been attached by some to the fact that during the so-called cholera typhoid a maculated, papular, erythematous exanthema has been observed, that may appear of decided urticarial character, or show a roseola-like appearance; it differs from the eruption of typhus in that it begins at the toes and spreads up to the trunk, where it is most manifest, becoming very imperfect on the face and head; the spots and papules may also run together and form a diffuse redness in different places—very much as is seen in certain forms of malaria. This eruption seldom manifests itself before the end of the first, and often not until during the second week, and notably most of the patients thus affected recover; it is not, however, so constant a symptom of cholera typhoid as to be pathognomonic, and is more apt to occur, perhaps, where sinapisms have been repeatedly or continuously applied to the extremities during the algid stage, or massage has been energetically used.

Sleeplessness, a condition of sullenness, etc., sometimes supervenes during apparent convalescence, when

the patients may either fall back into the condition of asphyxia, or continue to improve; but the latter is usually at the expense of numerous suppurations, manifested as a crop of boils, perhaps as abscesses of the parotid or of the larynx, or by general pyæmia.

It is needless to remark that this third stage is one of danger accordingly as it manifests in greater or less degree the pneumonic or typhic condition, though in any event it is apt to induce general impairment of the system that persists for a long period.

Of the anatomical changes that take place during or supervene upon a cholera attack, it is impossible to speak in complete detail. However, the great withdrawal of serum from the blood enables the bodies of those deceased to resist decomposition to a remarkable degree, hence the changes usually encountered post-mortem are lacking. It has before been remarked that the corpses are greatly shrunken, of dusky or livid color; that rigor mortis is rapidly developed and persists for an unusual length of time; and that very remarkable and violent contractions of the muscles are by no means uncommon, so much so as to give rise to weird tales of unfortunates buried alive, etc.*

Bodies of those who succumb six, twelve or eighteen hours after the attack, exhibit the same

*See pages 112, 113.

cyanotic appearance, and collapse of features, as in the last hours of life.

The circulatory organs and the blood exhibit the following conditions: When death is early, absence of the pericardial fluid is noted, or it is scant and sticky; later it is normal or slightly increased. The portion of the pericardium, which lies open and is attached to the outer side of the heart, *i.e.*, the visceral layer, is almost constantly the seat of ecchymoses, most numerous towards the base and posteriorly; it is rare to find on the parietal layer. Much more blood appears in the right heart than in the left—indeed the left heart is almost always empty,—which is apt to be of a pappy appearance, or exhibit soft coagulable and fibrinous clots, the latter gelatinous, or firm and colorless, either of which conditions may be present in the typhoid stage.

Lebert remarks he once found a fine fibrinous clot separated in the form of a membrane spread over the whole inner surface of the right ventricle; and that the perfectly soft dissolving clots which are often seen, correspond to no particular stage or condition. He also made a chemical examination of the blood of a patient dying in the typhoid stage, that revealed no increase either of urea or carbonate of ammonia as a constant condition, yet adds he would not “like to draw conclusions from these individual examinations.” Virchow admirably described the increase in the number of the white corpuscles in the heart clots.

As to the respiratory organs, they are seldom affected in their principal portions, though occasionally secondary diphtheritic and pseudo-membranous processes are encountered. The mucous membranes of trachea and bronchi are very much engorged with blood in cases of early death, and when there is moderate hyperæmia often covered with mucus in which are discovered more or less leucocytes; in exceptional cases the glands of the trachea are considerably swollen. Ecchymoses are not uncommon, but appear more frequently on the surface of the lungs, which latter are deeply engorged with blood, especially in their inferior and posterior portions, and often œdematous. Purulent mucus in the smallest bronchi, and the anatomical lesions of broncho-pneumonia and of typhoid-pneumonia, are conditions sometimes seen in cases of death—after three, five, or eight days; and pleuritis with sero-purulent effusion also belongs to these rarer complications; hæmorrhagic pulmonary infarctions are not infrequent.

The isolated œsophageal glands are often excessively swollen, the tube itself being cyanotic in the algid stage and ecchymosed at a later period. In his Zürich autopsies, Lebert often “found the epithelium detached, and once the lower part of the œsophagus covered with fibrinous diphtheritic membranes.” The stomach is distended and filled with colorless fluid in cases of early fatality, but later is empty and collapsed; when death occurs after the third or fourth

day it is apt to be filled with yellowish-green, sticky, gelatinous or mucous fluid; and the mucous membrane, at first hyperæmic, shows later numerous ecchymoses, and occasionally spots of bloody infiltration; when death occurs late, it is covered with abundant, tough, thick mucus, and perhaps spots of softening that probably are, in part at least, the effects of commencing decomposition.

The most marked changes, however, are found in the small intestine. Where the malady runs a rapidly fatal course, the intestinal peritoneum is dry, of a rosy color, or covered with a light layer of sticky fluid. In more prolonged cases the lesser bowel contains a greenish pultaceous mass, while the colon cœcum, etc., may harbor half-solid fæces; in the early periods the contents are of "rice-water" character.

During the attack proper, and immediately following, the glands of the small intestine are chiefly affected: Brunner's first, a condition that is constant; then the isolated and agminated glands, the former standing out in relief, their size varying from that of a millet-seed to a pea; Peyer's patches are granulated on the surface; swelling and engorgement most pronounced toward the ileo-cœcal valve. Aside from the hyperæmia and ecchymoses, the prominent glands give the surface a pale, milky, or yellowish appearance, and if the follicles are pierced they exude a whitish-gray fluid with fine granules and cell-nuclei, without leucocytes; the surface is smooth, for

the most part deprived of epithelium and villi, and the engorged glands admit of perfect artificial injection.

These typical changes are generally found in the first forty-eight hours; Lebert often observed the engorgement began to diminish at the end of from thirty-six to forty hours, though in some cases the glands continued infiltrated for four or five days, particularly if this condition persisted in the tissues immediately adjacent; as a rule, however, infiltration rapidly diminishes at the end of the second or in the course of the third day, when the glands present a flattened and somewhat wrinkled appearance, later becoming almost granular; they are still prominent, however, though shrunken in circumference, and of yellowish-gray (later almost slaty) color; occasionally a blackish-gray, brown, or brownish-red pigment is noticed, especially if ecchymoses have previously existed. In the second week, with rare exceptions, all infiltration and congestion disappears, though the glands may still continue thickened and abnormally colored. In the first period Peyer's patches are often found converted into a net-work, the follicles fissured as if ruptured; and as this condition is present in bodies twelve to eighteen hours after death, the supposition it is a post-mortem phenomenon is manifestly incorrect. In cases of early death, in some epidemics, the patches are ulcerated as in typhoid fever; the glands of the large intestine, also are found swollen,

prominent, and they likewise collapse at a later period, showing the same retrograde changes as those of the small intestine.

The mucous membrane between glands may share in the infiltration, and in the first stage is very deeply congested (almost cyanosed), generally ecchymosed or with extensive extravasations, so that great patches of mucosa are deeply ensanguined, a condition more frequently observed in the colon than in the small intestine; at an early stage also it quite often is softened, swollen, even œdematous, a condition that later is confined chiefly to spots; extensive softening of either the small or large intestine, however, is rare. The same changes in color, as noticed in the retrograde metamorphoses of the glands, occur throughout the mucous membrane, though less pronounced.*

The anatomical characters of secondary colitis of a diphtheritic or dysenteric nature, are seen comparatively often in some epidemics, in others are almost entirely absent. The mesenteric glands are quite often moderately swollen, but usually without much infiltration.†

“The spleen is in general small, rather wrinkled and shrunken, of good consistence, and moderately

*Lebert faithfully pictures all these details in his “Atlas of Pathological Anatomy.”—S.

†For the succeeding anatomical descriptions I must acknowledge my indebtedness to Herman Lebert’s monograph on the Zürich epidemics.—S.

supplied with blood, though sometimes seen enlarged in consequence of apoplectic effusion; in cases where cholera complicates typhoid fever, it is usually enlarged.

“The liver, in speedily fatal cases is often hyperæmic, and shows also numerous sub-peritoneal ecchymoses; at a later period is pale, marble-yellow or red, with isolated islands of fatty degeneration; the gall-bladder distended, in the first period with dark-brown bile, later of a bright-green color, semi-fluid, resembling mucus. Catarrh of the biliary passages, even of purulent nature, occasionally develops as a secondary affection. During the attack the bile is retained, but later, when again discharged, during a protracted convalescence or in the typhoid state, it seems to be abnormally constituted—a fact which makes chemical examinations at this period much to be desired.

“The bladder usually shows nothing abnormal; if death occurs in the first two days, it is contracted and empty. Still I have found in it, in exceptional cases, an ounce or more of cloudy albuminous urine, even after the disease has lasted from thirty-six to forty hours. Usually a little urine is found in the bladder in case of death on the third day, though often it may be empty even when death occurs on the fourth or fifth day. The mucous membrane of this viscus, in the beginning takes part also in the general cyanosis, but it is comparatively little marked and of little consequence.

“The kidneys may early take part in the disease processes, and even when death occurs in from sixteen to twenty-four hours, there is always observed an increase in the volume, and at the same time they are generally filled in both the cortical and medullary substances with blood in the form of stripes and punctated injections, and on the surface in star-shaped and marbled spots, with numerous and thick anastomoses; the superficial inter-canalicular vessels and capillaries of the Malpighian glomeruli also share in this congested condition, and ecchymoses are likewise not infrequent. Even in cases of death in the second half of the first day, the cortical substance of the kidneys is often found in an unmistakable condition of commencing decoloration, extending even from the surface deep down into the pyramids; also the capsules are frequently abnormally adherent. The microscope reveals at this early stage a remarkable epithelial proliferation in the urinary canaliculi, with cloudy swelling of the cell, the contents of which (consisting of numerous albuminoid granules) may be dissolved by acetic acid. Now and then may be discovered, as early as at the end of the first day, distinct transparent cylinders in the interior of the urinary canals. The kidneys, therefore, are decidedly affected on the very first day of a pronounced attack of cholera.

“In the course of the second day I have noticed either the same commencing decoloration, or more

marked changes. The hyperæmia is now either confined to spots, or general with simultaneous decoloration of the canals and cortex; casts are present in great quantity—pressure empties from the papillæ a cloudy albuminous urine containing them, and not infrequently crystals of uric acid. The mucous membrane of the calyces and pelves is usually hyperæmic, with injection of the fine vessels, and the microscope reveals progressive degeneration of the cells. In the course of the third day the decoloration is so far increased as to involve the whole cortex, and granulations are present; the blood seems to be very unequally distributed; the surface, uneven, rough, and closely adherent to the capsule. The cells continue to be detached, the development of casts proceeds, the fatty elements increase and now show themselves as granules and oil drops in constantly increasing quantity in the epithelium and outside of it, in the interior of the canaliculi, and in the casts.

“These alterations increase in the typhoid stage as well as during imperfect convalescence. The kidneys, according to many accurate measurements, are from one-sixth to one-third larger than normal, the granulations more abundant, and the decoloration advanced to the pyramids and even between them. The substance of the kidneys is now softer, more easily torn, and infiltrated with a dirty yellow, fatty, and albuminous fluid. The mucous membrane often

seems thickened. In cases where death occurs late, all the signs of resolution are present; in favorable cases, all these seemingly grave lesions quickly disappear, and the kidneys regained their normal condition. Strange to say, chronic nephritis caused by or incidental to cholera, is rare" (Lebert).

The chemical examinations of the various organs undertaken by Staëdeler, in Zürich, in 1855, yielded no special results. Leucin was found in the liver, and small quantities of uric acid in different organs. In the spleen, leucin was once detected; in other cases inosite, uric acid, and much pigment. The kidneys contained comparatively a great amount urea, some leucin, bile-pigment, and uric acid, but no inosite.

Glancing now at the whole duration of cholera: For the period of incubation may be allowed from five to seven days, often much less, sometimes longer. Where prodromata exist, their average duration is from one to three days. Next comes the stage of attack, which is the second, or (when prodromata are lacking) at times the first stage; this may prove fatal, in from six to eight hours, or even less, but varies in fairly severe cases from twelve to twenty-four hours.

With the end of the attack proper (the second stage) comes the period of reaction, usually at the end of from eighteen to twenty-four hours, and now the patient either dies from cyanotic asphyxia, or the reaction is perfectly established and the real cholera

is happily and definitely over. The stage of reaction may pass into speedy convalescence, which may terminate in the second half of the first week; or the convalescence is protracted, either without further critical symptoms or with transition into the typhoid stage. The typhic stage, in turn, may lead to fatal or fortunate termination in the last days of the first, or in the first days of the second week of the whole duration of the disease. It is a rare exception for patients to die of cholera after ten or twelve days, or to be affected with long-continued, bad sequelæ, though a weak invalid condition may persist for a long time as the result of defective nerve action.

Finally, it must be remarked that it is impossible, no matter what the scope of the work, to describe all the sequelæ and complications of cholera; where the circulation is so profoundly disturbed, and the nerve toxæmia so virulent, the most multifiform local congestions and inflammations are possible.

CHAPTER VII.

CHOLERA DIARRHŒA AND CHOLERINE.

The mildest form of cholera is that simulating a simple diarrhœa, in which the evacuations follow each other more or less closely, are very copious and watery, but *not* altogether devoid of consistence and color, and retain in some degree the characteristic fæcal odor; they perhaps are not accompanied by colicky pains or tenesmus, and cause no constitutional or other disturbance except a moderate degree of depression and relaxation. Such frequently do not appear in official lists, but, as Niemeyer pertinently observes, "though not considered by the police as cholera, they should be so recognized by science. This is shown:

(1) "By the larger number of cases of diarrhœa occurring during cholera times, although almost all sensible people carefully avoid errors of diet, catching cold, and other sources of injury:

(2) "The great obstinacy of these cases:

(3) "The well known transportation of the disease by persons suffering therefrom:

(4) "Especially by the numerous transformations of simple cholera diarrhœa into the most severe form of the malady.—Many patients, especially of the poorer classes, worried by a diarrhœa which will not give way to domestic remedies, go to the doctor for a prescrip-

tion at noon, and in the evening lie cold, pulseless, and cyanotic, almost in a hopeless state. . . . I deem it much more important to determine the frequent occurrence of gradual transformation from simple cholera diarrhœa to so-called cholérine, and to malignant cholera, and to prove the identity of these three forms, than to seek for pathognomonic signs of epidemic cholera.”*

Cholera diarrhœa and cholérine, which constitute the milder forms of the disease, during the prevalence of epidemics also present varied phases, and more than all varied characteristics in different epidemics. Lebert, Zehnder, Karl Liebermeister, and others, have noted how greatly personal predisposition favors or inhibits, as the case may be, extension or repression; that an imported case in any one locality may be confined to a single individual, to a single house, even to single room in a dwelling, while at other times one case may be the focus of a raging pestilence. Thus the history of different epidemics, particularly in large cities, shows the greatest variety

* “Text Book of Practical Medicine,” Vol. ii. New York, 1884.

* How probable it is that a swiftly fatal attack is developed from profound toxicity of the central nervous system through the medium of the absorbents, especially those of the portal area, by an excess of the cholera poison, is shown by phenomena developed in acute arsenical poisoning, which has several times been mistaken for true cholera (see p. 107).
—S.

of effect, accordingly as the cholera poison finds conditions for development more or less suitable. Again, it has been observed, as a rule, that prodromic diarrhœas are more frequent and more widely diffused in malignant and extensive epidemics than in those of less extent. Yet Lebert declares,† in the great epidemic in Paris of 1849, which is computed to have claimed something like ten thousand victims, premonitory intestinal flux was wholly absent in from five to ten per cent. of the pronounced cases; in Zürich it was absent in 33.3 per cent. of pronounced cases; and that absence or presence of prodromic diarrhœa had no apparent influence in determining the ultimate result of the attack, as the recoveries and deaths were about equally proportioned in regard to this symptom. Also, he adds, "I found that the prodromic diarrhœa was absent in seven-eighths of the cases of true cholericine (with colored stools). In Paris as well as in Zürich and Breslau, in 1866 and 1867, I saw a number of cases of diarrhœa which were due to the influence of cholera, recover without treatment and without subsequent cholera. On the other hand, in July and the beginning of August, 1866, I witnessed such obstinate and violent cases of cholera diarrhœa in the Breslau garrison of cuirassiers, where I had charge of a large ward of wounded patients, that it required the utmost effort to prevent an outbreak of cholera."

† Ziemssen's "Cyclopædia of Practical Medicine." New York, 1874.
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There is no marked distinction between common intestinal catarrh and cholera diarrhœa, yet the latter presents certain definite peculiarities which are entitled to consideration in seeking a definite diagnosis.

Cholera diarrhœa is usually sudden and unexpected: As already remarked, is apt to be excited, or receive its ultimate development, through "taking cold" or errors of diet; and to *intemperance* in the habitual use of alcoholic beverages more than any other single cause may the proclivity to this form of flux be ascribed.

Loss of appetite, thirst, indigestion, are either present or absent in only slight degree; patients generally, however, complain of an excessive feeling of lassitude—are extraordinarily weak, feeble and uncomfortable,—and not unfrequently exhibit despondency, out of all proportion to the frequency or quantity of the flux.

There is nothing specially peculiar about the stools other than they are simply fluid, as in any diarrhœa; often there are but two or three discharges during the twenty-four hours, more rarely from six to eight, and are more apt to supervene during the night, and to be accompanied by distressing rumblings and gurglings.

In some cases the diarrhœa persists only a few hours or days; in others, one or two weeks, with considerable intermissions, during which the individual feels perfectly well. Sometimes it regularly intermits or

remits during the whole course of the epidemic in that particular locality, and ceases only as the latter disappears. Even in the milder cases of cholera diarrhœa may be sometimes observed individual evidences of cholera proper, such as suppression of urine, light cramps in calves, colorless and odorless “rice-water” stools, that are merely of temporary duration, and are more generally characteristic of cholérine. Neither is it a rare occurrence for a cholera diarrhœa, which remains in other respects without danger, to suddenly increase in virulence, with violent, quickly repeated stools, as in true cholera, and yet cease quickly and be followed by prompt recovery.

Cholérine, as the term indicates, is a lesser cholera; in other words, it is a condensed picture of the mildest form of the more grave attack, and may, indeed often does, present some of the serious symptoms peculiar to the latter. It is usually ushered in by such prodromal symptoms as malaise, headache, diminution of appetite, muscular or general physical weakness, of at least twelve and often twenty-four hours' duration; there is also general restlessness, insomnia—or at least the hours of sleep are productive of discomfort rather than rest; and the attack culminates in the middle of the night or early morning by sudden awakening due to demand for stool. Now is observed a copious yellowish-brown, almost watery discharge, with characteristic diarrhœic odor, but succeeded by a second still more fluid, and others

again at brief intervals to the number of three, six, eight, twelve or more, until—when they become very numerous—there is gradually less and less evidence of odor, color, or form, and they assume a decided “rice-water” character.

With the first evidence of diarrhœa, nausea is apt to supervene which, after repeated stools, is merged into emesis, the ejected matter being first of a yellowish green hue with intensely sour bitter taste, very fluid in character, finally, perhaps, colorless, whey-like, showing a deposit very like grains of bruised rice; after a few times, the quantity is lessened, the act itself becomes more infrequent, and finally it ceases altogether after a few hours; this vomiting is not at all painful, retching being a comparatively rare concomitant, but the fluid pours forth in a manner resembling an easy act of regurgitation, though its profuse quantity is very suggestive of its choleraic nature. The sufferer in the meantime becomes very much reduced; but with the cessation of vomiting and diarrhœa, either recovers very rapidly, or temporary typhoid symptoms manifest themselves. While complete convalescence may be established in a few days, it is often a matter of two or three weeks. In the favorable cases of cholera, convalescence occurs in two, three or four days.

Sometimes the inclination to diarrhœa lingers for some days after the convalescing stage has set in, with all the concomitants of anorexia, borborygmus,

occasionally twinges of colic, etc.; not infrequently also there is continued nausea, especially after the ingestion of food; and a distressing tendency toward cramping of muscles of legs (calves) is more or less apparent according to the severity of the attack. Lebert remarks he has also seen moderate cooling of extremities; that he personally experienced almost complete aphonia for twenty-four hours after a seizure, and did not fully recover his voice until "at the end of several days;" also noticed considerable reduction in the quantity of urea, as well as the temporary occurrence of albumen and casts in very dark scanty urine. It is perhaps needless to say that gastric catarrh not infrequently interferes with convalescence; that errors in diet may lead to fatal relapse; and that the temporary typhoid state, when it supervenes, is manifested by such symptoms as headache, vertigo, roaring in ears, cloudiness of vision, great debility, sopor, etc.

It is also of interest to remember that where this "little cholera" is seen in houses and families, frequently in the course of a few days a malignant asphyctic cholera will develop, and that it therefore may lead to genuine and fatal cholera when the individual (or individuals) is sufficiently receptive toward the poison; that cholerine may itself prove fatal in weak and aged patients; and when cholera prevails in a large city with great severity, the number of persons attacked with real cholera is always relatively

smaller than those attacked with cholera diarrhœa and cholerine. Moreover, cholera diarrhœa and cholerine occur much more frequently in places some distance from the centres of the disease, while true cholera prevails at these centres. In all these cases it is probably the diminished capacity of the surroundings for infection accounts for the relatively greater dissemination of the lighter forms of the disease; but, as just remarked, these lighter forms by rapid multiplication, or increased virulence of the poison of the disease, under favorable conditions of the organism, may lead to dangerous attacks of cholera.

In enumerating these phases of the epidemic more specifically, it must be understood that in differentiating the three forms there is no recession from the position I shall, in a later chapter, endeavor to establish that all are *de facto* cholera. And, further, it may be added, it is a matter of great personal doubt whether *any* cholera is derived from a special germ or poison, other than that resultant upon any common decomposition; or possibly that the Asiatic form is due to the common poison supplemented by an imported one, that after all is but the same intensified in its original habitat and by transmission; or, again, that all cholera is due to a poison of common decomposition that was foreign originally, but since importation has become in a sense acclimated. There are few medical men who have not seen cholera nostras—or as it is termed in

this country, sporadic cholera, or cholera morbus—with positive “rice-water” discharges, colorless and odorless, outside of the period of any epidemic; I have repeatedly encountered such in my own experience, and in the service of others; and Lebert, J. M. and D. D. Cunningham, Bouchard, Leiter, Zehnder, Fayrer, Niemeyer, Trousseau, Semmola, Tanner, Sir Wm. Aitken, and a list who are fairly “Legion,”* corroborate, and have also expressed the opinion cholera infantum is only cholera sporadica, *modified by age*.—*A’ propos* of this it may be mentioned that Chas. Talamon† has recently adduced considerable evidence tending to demonstrate the unity of these maladies :

He points out that ravages of cholera diarrhœa, of epidemic nature, occur from time to time without any evidence of a foreign or imported factor, in localities in which true cholera has, sometime or another, manifested itself; and likewise reference is made to a recent epidemic, the exact prototype of another occurring in 1866, in the neighborhood of Paris, clinically diagnosed as cholera nostras, but which on investigation in no way differed pathologically from the so-called Asiatic malady. The cholera bacillus was demonstrated in the dejections also.

*See “Nature and Treatment of Sporadic Cholera” by Alex. Harkin. W. Renshaw, London, 1885.—S.

† La Médecine Moderne, 1892.

CHAPTER VIII.

SPECIFIC PATHOLOGY.

I have been thus particular in enumerating the general outlines and peculiarities of this disease, which presumably are familiar—or, at least, they should be,—to every medical man, in order to more forcibly call attention to their physiologico-therapeutical relations. It has already been remarked, the neurotic character of the disease is most apparent, and that there is no relation between the quantity of fluid lost by the bowel and stomach and the malignancy of the onslaught.

The simple diarrhœa which ushers in an attack, or that may constitute almost the only manifestation of the malady, is especially remarked upon by all observers for its stubborn character; it yields to none of the customary remedies, and is influenced scarcely at all by opiates or astringents. This presupposes a lesion deeper than, and behind, the visceral and thoracic organs that, apparently, are most involved, and one has no option but to refer the same to the *central nervous system*.

In 1870 M. F. Moreau, and also S. G. Vasquez, demonstrated by a series of observations,—corroborating those of Edward Pflüger and Otto Nasse, and since repeated by many others,—the influence of the sympathetic nerves upon the intestinal canal,

which influence is most pertinent as evidencing the source and character of the watery diarrhœa in cholera, and likewise its sudden occurrence without any form of premonition or warning.

Both found when these nerves were divided, the portion of the intestines segregated by the operation rapidly filled with clear alkaline fluid, colorless and slightly opaline—except as, at the outset, they might become mixed with material still remaining within the gut,—which precipitated flocculi of organic matter on boiling.* Transudation of watery fluid into the intestines, due to capillary diffusion, takes place as the result of paralysis of the sympathetic; the occurrence of sudden hyperæmias, transudations, and ecchymoses, in some thoracic or abdominal organ, may have a neurotic basis.†

Paralysis or hyperæsthesia of the sympathetic—conditions that differ physiologically *only in degree*,‡—induce vomiting, retching, etc. When the nerves are divided below the solar plexus, which is situated in most intimate relation with the splanchnic area, the

* *Vide* Flint's "Physiology of Man." New York, 1874.

† *Vide* Carpenter, Dalton, Draper, Kirk, Paget, Landois and Sterling, Foster.

‡ There are several circumstances that support this view, chief of which are, the association of pain with both hyperæsthesia and numbness, especially in highly sensitive parts, and the difficulty of differentiating the condition producing the one from the other. "It is clear," says Dr. C.

secretion of urine and urea sinks quickly to a minimum, and may perhaps cease entirely, as shown by Coni Peyrani, who is corroborated by Brown-Séquard and Claude Bernard. These latter authors, moreover, further observed that partial segregation of the sympathetic below the inferior cervical ganglion induced marked depression of temperature, made more manifest toward the surface and in the cutaneous area, but succeeded by rapid *increase* of temperature after death. "Paralysis of vaso-motor nerves decreases temperature," says James Kirk; and the sympathetic pertains to the vaso-motoric system.

Lebert declares that while the most important and constant anatomical clinical localization of the disease occurs in the small intestines, there "is a possibility the often so violent discharges of serum are incited by the central nervous system, the excitement originating in the *vaso-motor centres*." Again: "The cerebro-spinal fluid is entirely absent in cases where death occurs at an early period, or is present only in slight quantity and of almost pasty consistence. But I have seen it more copious when death has occurred

Handfield Jones, "there is no opposition between them: all are present together. Now paralysis, numbness, anæsthesia, hyperæsthesia are evidently failure of functional power; and of the same import is the occurrence of various degrees of paralysis, or of paresis, paræsthesia, analgesia, which may be, and are, associated as analogous affections of the motor and sensory nerves."—S.

at the end of thirty-six hours; and, where death occurs still later, it may even exceed the normal amount. The pia mater loses its marked hyperæmia in a more protracted course of the disease, and becomes dry, perhaps icteric. The fluid of the ventricles remains scanty even when death occurs at later periods.

* * * Ecchymoses of the external surface of the brain (pia), or on the internal surface (ependyma) are not rare. There may be capillary effusion into the pons, and once I saw a fresh effusion of blood between the dura and arachnoid."—All this is certainly suggestive.

Again, it must be borne in mind that while "rice-water" stools accrue to all choleraic attacks, except the most pronounced and violent form known as *cholera asphyxia*, *cholera siderans* or *cholera sicca*, they are, *per se*, by no means pathognomonic, but result always from influences of the most powerful character brought to bear upon the central nervous system. They are a concomitant of terror and mental perturbation; of heat apoplexy; of arsenical poisoning;* of autogenetic

* How probable it is that a swiftly fatal attack is developed through nerve irritation manifested in a portion of the small intestine, is shown by the action of arsenic, which may induce fatal poisoning in a few hours, with symptoms perfectly resembling those of cholera. Late in the autumn of 1854, a woman who had been suddenly taken sick during a criminal trial, entered Lebert's Clinic in Zürich. She died in hospital after a number of violent, perfectly colorless,

toxication by specific ptomaines; of toxication by certain alkaloids, etc.; and, moreover, the dejections, as in cholera, hold a profusion of material commonly supposed to be intestinal epithelium (but which is really the result of transformation taking place in the effusion of blood plasma), and likewise contain serum-albumen, mucin, and a large amount of salts, chloride of sodium preponderating to such an extent as often to exceed in amount all organic matters.* But the blood in cholera, as an almost invariable rule, is free from bacteria, either actual or potential; this is the case as well shortly after death as during life, and holds in regard to every stage of the disease.

Further, choleraic, arsenical, and alkaloidal poisonings, of a particular class, likewise thermic apoplexy, alike induce a high specific gravity of the blood—an

very copious discharges. The small intestine showed an immense accumulation of colorless fluid, and clinical examination of the contents of the stomach revealed unmistakable evidences of arsenical poisoning. Lebert further adds: "In the summer of 1847 I was told by Louis, in the Hotel Dieu, that the Duke of Choiseul, who had been arrested the day before for the murder of his wife, died suddenly of cholera, and he wondered at it greatly, because cholera was nowhere prevalent. Louis was the Duke's physician and, as is well known, one of the greatest diagnosticians of our time; nevertheless it soon turned out the Duke had poisoned himself with arsenic."—S.

* The proportion of solid constituents in all, varies from 1.20 to 2.40.—S.

average of 1.0701, against 1.0503 in health,—which is remarkably tough and viscid, the corpuscles increased in number but abnormally impoverished as to salts; the amount of fibrin is unaffected, but the serum is very dense, extremely rich in albumen, and contains more phosphates and potash salts (though less collectively) than normal; moreover, contains some urea, together with extractive matters that seem to possess the quality of rapidly converting the former into carbonate of ammonia (Day, Hoppe-Seyler, C. Schmid). Draper also remark: “In cholera the constitution of the blood is so changed that the cells can no longer carry oxygen into the system; the heat-making processes are put a stop to, and the temperature declining, the body becomes of marble coldness characteristic of this terrible disease,”—phenomena that accrue to *muscarine* and certain other alkaloidal poisonings.

Armand Trousseau says the varied and peculiar symptoms exhibited by asphyctic cholera, can be explained only by referring to a poison having specific effect upon the nervous system; and Wilhelm Erb adds,* referring to poisons as a class: “They cause lasting paralysis, as a rule, only when their action is slow and repeated, more rarely when they are acute; they cause the most varied forms of paralysis and paraplegia, from simple weakness and paresis to com-

* Ziemssen's “Cyclopædia of the Practice of Medicine.” New York, 1874.

plete paralysis, . . . with or without disturbance of sensibility.

One of the functions of the sympathetic, if the deductions of Jno. W. Draper maybe accepted, is "the equalization or balancing of nerve force, storing up all transient excesses, and providing for all transient deficiencies."

This sympathetic nerve system, too, transmits sensations so tardily that the economy may be violently, even fatally poisoned through the central nerve organs, long before the customary effects are manifested by the usual symptomatology. Says Jno. C. Dalton :* "Evidences of sensibility are much less acute than in other nerves, and show themselves *only after prolonged application of the exciting cause*!"

Damp, cold, malaria, chorea, and certain poisons, all act in the same precise way by deranging molecular nutritive actions of the nerve structure, and so unfitting the latter to fulfil its function : and anæsthesia and hyperæsthesia are alike failures of functional power, varying only in degree. Again hyperæsthesia and irritation may be the result of dual action, viz : Of poisoning of cerebral centers, and of local irritation.

The foregoing most certainly sheds new and most welcome light on the pathology of cholera, especially the frequent steady march to fatality during moments

* "Human Physiology." New York, 1890.

that promise so much in the way of amendment as to mislead the most astute observer.

And herein perhaps lies the arrow that fatally pierces the joints of our therapeutic armor, when is sought to oppose the disease by treating that which is most apparent—the local manifestation; the evil has been wrought long before its faintest symptomatology is made appreciable to human understanding, and consequently is apt to be beyond the power of any method or theory of antagonism or revulsion, however perfect. In cholera, then, often the patient may be *fatally poisoned* before the presence of the disease can be detected or even surmised.

In *facies cholericæ* is observed a condition that may be duplicated by segregation of the superior cervical ganglion of the sympathetic, or by its extirpation; the eyeball is drawn back into the orbit, causing partial closure of the upper and lower lids and flattening of the corneæ; the countenance becomes withered and ghastly; in short, is brought about a cadaverous aspect that sometimes precedes death in long-standing disease, but here supervenes in an hour or two.

When death occurs during the invasion of the disease or in the stage of collapse, in the more marked cases the appearances, as previously shown, are: The bodies remain warm for some time—the temperature may rise after death, perhaps even to 103° Farh., and so continue for several hours (note the observations

of Brown-Séquard and Claude Bernard, before cited); the rigor mortis* soon appears and is extended over a preternaturally long period of time; the muscles (particularly of hands, arms, and legs) sometimes exhibit a peculiar spasmodic twitching before rigor mortis sets in, so much so that the Cholera Gazette for 1832

* Professor A. Paltauf read a paper before the Association of German Physicians, at Prague, on some experiments made to show the causal connection between rigor mortis and deaths from poison. For the purpose of these experiments such poisons were used as were known to exert a certain influence on the muscular system, either by acting directly on the muscle substance, or indirectly by affecting the nervous system. Amongst the poisons belonging to the first series, curare always considerably *delays* the occurrence of rigor mortis. Amongst those acting on the central nervous system, strychnine, picrotoxin, camphor, and the salts of ammonium and arsenic, *accelerate* the occurrence of rigor mortis. The acceleration is still more increased by artificially prolonging the stimulation of the muscular system, but is again arrested on the occurrence of paralysis. Veratrine and physostigmine cause only a slight acceleration of the rigor mortis, but with caffeine and its chemical derivatives—the rhodan salts—this acceleration becomes considerable. To study the influence of the nervous system at the time of occurrence of the rigor mortis, Paltauf divided the nerves and the spinal cord, with the result that the more a muscle had been stimulated by the poison the sooner was the rigor mortis observed, independently of its connection with the spine, if such connection existed. The reaction of the rigid muscles was in the case of many poisons, as has been generally believed, acid. Other poisons, however (such as camphor, ethyl-theo-

declares the soldiers were accustomed to bind the limbs of their dead comrades to the bed-frames in order to prevent shocking the more timid of the living.* All these manifestations point indubitably

bromine and the rhodan salts), gave, contrary to the general assumption, an alkaline reaction. This alkaline reaction affected, however, only the anterior portion of an animal in which, after the poisoning, the cord had been divided. The posterior part of the animal, in which the rigor mortis was delayed, showed the usual acid reaction until the alkaline reaction of putrescence took place. Where the reaction of the anterior portion of the animal was alkaline it often became, after the reduction of the rigidity had passed off, neutral or slightly acid before putrescence once more made it alkaline. Division of a single nerve had the same result, and it was possible to cause either alkaline or acid reaction of the various muscles of one extremity by respectively leaving the nerve entire or by dividing it. Paltauf also approached the solution of the question of the existence of a cataleptic rigor mortis. He found that the convulsive muscular contractions of an animal poisoned by camphor and suddenly killed by strangling led to immediate rigor mortis, and he therefore believes in a cataleptic rigor mortis.—*Weiner Medicinische Presse*.

Some time previous to this paper, Prof. Paltauf announced he had observed "the greater the hyperæsthesia of the sympathetic, the greater the rigor mortis; but if the sympathetic had only attained a condition of paralysis, the rigor mortis was very greatly delayed."—S.

*Bodies of those who died from cholera, on resurrection after burial were sometimes found turned and twisted in their coffins; and this accrues often to those that have suc-

to poisoned nerve centres, being phenomena especially apt to follow upon specific toxic fatality. And long since T. Lauder Brunton recognized the symptoms of cholera were precisely paralleled by those of muscarine poisoning. Muscarine, though an alkaloidal product of *Agaricus muscarius* (fly agaric), is also a ptomaine, and consequently of animal as well as vegetable origin, and one of the most powerful nerve-poisons known. Its specific action is upon the sympathetic, inducing either paralysis or hyperæsthesia according to the degree of toxicity; it induces, as just remarked, all the phenomena of cholera, even to stoppage of the heart in diastole, and reflex derangement of kidneys with suppression of urine; moreover, its toxic manifestations are exceedingly dilatory for the same reason.

Another point of moment to be recalled is that the functions of the sympathetic, which belong to the vaso-motor group, are adjuvant to the cerebro-spinal system.

Carpenter appears also to have had a partial insight into the workings of the sympathetic, for he says:† The nerves of this system—"in which tubercumbed to other specific poisons, as the result of decomposition and stimulus of nerve centres by the formation of cadaveric alkaloids that seem to have the power of inducing in dead tissue the same phenomena that result upon galvanic stimulus.—S.

† "Principles of Comparative Physiology." London, 1854.

lar fibres derived from the cerebro-spinal system are combined in various proportions with those gray or organic fibres which have their centres in the proper sympathetic ganglia,—possess a certain degree of power of exciting muscular contraction in the various parts to which they are distributed. Thus by irritating them, contraction may be excited in any part of the alimentary canal from the pharynx to the rectum, according to the trunks that are irritated; in the heart after the ordinary movements have ceased; in the aorta, vena cava, and thoracic duct; in the ductus choledochus, uterus, Fallopian tubes, vas deferens, and vesiculæ seminales; and the very same contractions may be excited by irritating the roots of the spinal nerves from which the sympathetic trunks receive their white fibres; and there is strong reason to believe that the *motor* power of the latter is entirely dependent upon the cerebro-spinal system. That even the *sensory* endowments the sympathetic trunks possess, are probably to be referred to the same connection. The parts exclusively supplied by the sympathetic trunks do not appear to be in the least degree sensible; and no sign of pain is given when the sympathetic trunks themselves are irritated. But under certain diseased conditions of these organs, violent pains, are felt in them; and these pains can only be produced through the medium of fibres communicating with the sensorium through the spinal nerves. . . . There is much reason to believe, however, that it (the

sympathetic system) constitutes the channel through which the passions and emotions of the mind affect the organic functions; and this especially through the power of regulating the calibre of arteries. We have examples of the influence of these states upon the circulation in the palpitation of the heart which is produced by an agitated state of feeling; in the syncope or suspension of the heart's action which sometimes comes from sudden shock; in the act of blushing or turning pale, which consists in the dilatation or contraction of the small arteries (arterioles); in the sudden increase of the salivary, lachrymal, mammary, gastric and intestinal secretions under the influence of particular states of the mind, which increase is probably due to the temporary dilatation of the arteries that supply these organs.* It is probable that the sympathetic system not only brings the organic functions into relation with the animal, but also that it tends to harmonize the former with the latter, so as to bring the various acts of secretion, nutrition, etc., into mutual conformity. The distinctive functions of the gray or organic fibres, and of their ganglionic centres, constituting the proper visceral system . . . not improbably have some direct influence upon the chemical processes which are involved in such changes, and may thus affect the *quantity* of such secretions; whilst the office of the tubular fibres may be rather to

*Also in the enuresis and diarrhœa that supervenes upon fright.—S.

regulate the diameter of the blood-vessels supplying the organ, and thus to determine the *quality* of their products.”

At the risk of prolixity, since the point to be made is of the utmost importance, I may repeat it is essential to the better understanding of the pathology of cholera to note that the phenomena peculiar to the malady, and the symptoms and manifestations relied upon for both ante-mortem and post-mortem diagnosis, are conclusive evidences of its neurotic origin, and of profound toxicity by some agent closely allied to certain alkaloids and acting upon the central nervous system, fatality being hastened by predisposing causes that induce depression of such nerve centres.*

* Toxic alkaloids as the cause of many cases of accidental poisoning have come into prominence of late years, and every summer instances of ptomaine poisoning are reported. An interesting article on this subject appears in the “Boston Medical and Surgical Journal” of August 4th, 1892. Much remains to be discovered relative to the kinds of ptomaines that may develop in both animal and vegetable substances out of the body, as well as of the toxins that may form in food after its ingestion. Doubtless the possibilities of ptomaine formation are very great, and under unusual conditions of insalubrity—hot, damp weather, sewage emanations, etc.,—the work of decomposition may go on with extraordinary rapidity, and, under such influence, tox-albumens of great power may form in food that to the eye and taste is still wholesome. There is accumulative evidence to show that this is so. . . . Cases

—That we do not know the poison specifically, is of little matter; neither do we know whether this poison is developed external to the economy, or induced within the body through miasmatic, bacillar, telluric, meteorologic or physiologic metabolism:

1. Watery diarrhœa and vomiting, both fluids being odorless and colorless:

2. Tetanic convulsions or cramps, followed by muscular flaccidity and lack of cutaneous sensibility and elasticity:

3. Diminished respiration; spasmodic contrac-

of poisoning by food of a relatively innocuous character are not uncommon. Some portion of the ingesta becomes an irritant poison, a fit of indigestion ensues, and often the offending substance is speedily expelled by vomiting. The matter of idiosyncrasy need not detain us here; it is known that certain articles of diet, such as cheese and shell-fish, are toxic to some persons. Generally the state of the alimentary canal at the time is responsible. The most common and best known of the various forms of indigestion is that in which, from the absence or the deficiency of gastric juice and other digestive fluids, the alimentary bolus becomes a gastro-intestinal irritant, provoking vomiting, purging, and a catarrhal condition of the digestive tract. A second stage in the process of acute indigestion arises from the presence of abnormal fermentations and decompositions in the alimentary canal. The food substances break up into organic acids and alkaloidal products of a lower order, which are in part absorbed and produce constitutional disturbances. This stage borders very closely on that of ptomaine formation. There is good reason to regard cholera

tion of circular fibres of bronchi, proximate and ultimate; flagging circulation, with more rapid heart-beat:

4. Depression of temperature below normal—perhaps from five to twenty and more degrees:

5. Suppressed urinary secretion without sufficient pathological manifestation—manifestly due to powerful reflex causes:

nostras and the gastro-intestinal catarrh of infants as kinds of ptomaine poisoning due to multiple causes, of which weakening of the alimentary canal and consequent poverty of digestive fluids, the ingestion of food of an indigestible character, the putrefaction of the latter and the formation of toxins, are the principal factors. That a considerable number of persons may simultaneously be attacked with illness after a large meal, owing to the influence of causes such as have been above mentioned, cannot be a matter of serious doubt. Similar conditions of faulty hygiene produce, in individuals with similar organic susceptibility, results essentially the same. A remarkable case of this kind occurred in America on July 23d of this year. Out of a total of seventy persons who dined together, about fifteen became more or less violently ill within a few hours, exhibiting the symptoms of cholera. The general opinion, as advanced by physicians who had opportunities of studying the facts is, that the sufferers were persons who had eaten rather immoderately of indigestible dishes; that the hot weather, the bad air, and the bad water of the place, co-operated in bringing about the necessary predisposition in the guests, and that therefore the "Salisbury sickness" was a local and limited epidemic of cholera nostras.—*The Lancet* (London), Sept. 17th, 1892.

6. Intense thirst—an evidence of functional disturbance and vicarious nourishment:

7. Immediate disorganization of blood corpuscles, similar in character but slower in degree to that supervening upon serpent poisoning:

8. After death an abnormal persistent increase of temperature, reaching 103° Farh. perhaps:

9. Speedy post-mortem desquamation of epithelium:

10. Post-mortem spasmodic muscular contractions such as follow upon galvanic stimulus:

11. Speedy rigor mortis persisting for an abnormally long period, and in proportion to the hyperæsthesia of the sympathetic:

12. The most virulent symptoms may usually be mitigated by antagonizing the sympathetic:

13. One attack of cholera seems to protect the individual against a second, in greater or less degree, since recurrences of the malady are not frequent:

14. Everything that induces shock (a purely nervous condition), or reduces muscle and nerve vitality—the two being in a measure physiologically synonymous and inter-dependent—increases predisposition:

15. Other epidemics, as influenza, that have a depressing effect upon the nervous system, promote cholera.—All other diseases during cholera epidemics become more aggravated and more fatal :*

*See observations on pages 10, 11 and 127.

16. Severe attacks of cholera are especially apt to follow wine suppers and debauches of all kinds—from depressed or deficient nerve tone:

17. Fear is often a prime factor in promoting the spread of the malady.—Though Lebert thinks this has obtained undue evidence, because “the greater the fear, the more minutely are all preliminary measures carried out.” Manifestly Lebert’s proposition is more true in abstract than in reality.

CHAPTER IX.

PROPHYLAXIS.

The cause of cholera has been shown, then, to be some morbid agent exercising a toxic effect through the central nervous system. The precise nature of the agent is unknown, but its effects are only too apparent; and experience has taught that it attacks the poor in a much larger proportion than the rich, the unclean rather than those practicing sanitation and the laws of hygiene.

It is also well known that in proportion to the prevention of distribution of water fouled with sewage, and to the removal of destitution, filth, foul air, and other great factors of disease, so, generally, is destroyed the agency through which the cholera poison operates.—Thos. Hawkes Tanner, especially remarks the lesson taught by the epidemics of 1853-54 and 1865-66 in England, viz., that even poverty-stricken denizens of an unhealthy neighborhood, supplied with *pure* water, are more certain to escape than the wealthy residents of fashionable parks and squares when the latter consume bad water. Still, that overcrowding is an important factor in disseminating the malady—probably through re-consumption of exhaled gases of respiration, tending to poisoning of the circulation and central nervous system with carbonic oxide—is not to be denied. Within the walls of an

establishment for pauper orphans at Tooting, England, in 1853-54 there were assembled 1,395 children, little more than one hundred cubic feet of breathing space being available for each child, against a requirement of 1,500 (and 500 is the very smallest compatible with health). Here, in a single night, the epidemic seized sixty-four of the inmates, 300 being laid low within a week, during which time 180 died. Again, in the workhouse at Taunton, with 276 occupants, and with breathing space in many of the rooms not exceeding sixty-eight cubic feet for each person, cholera swept away nearly twenty-four per cent. in the brief period of six days; while in the gaol of the same town, where each prisoner had a breathing space of nearly 900 cubic feet (and in some instances more), not a single case of cholera or diarrhœa occurred!

When an epidemic is prevalent, there are also certain conditions other than filthy surroundings, bad water, and foul air, that render individuals liable to the disease, such as unwholesome, indigestible food—stale meat and fish, game that is “high,” butcher-made sausages (since these latter are largely worked up from scraps and unsold remnants at the spoiling turn), withered vegetables, over- or under-ripe fruit, etc. Other predisposing causes are vitiated *damp* air; exposure to all forms of miasm; intemperance; insufficient protection from cold and inclement weather; excessive fatigue; long abstinence from food; diar-

rhœa; in fact all irregular habits, including loss of sleep, excessive venery, *et al.*

Hence the old maxim of an ounce of prevention *vs.* a pound of cure, in this malady receives apt illustration. To secure immunity it is essential to form regular habits of life—to live by rule, in a measure, for sudden radical changes, even if for the better in a general way, are sometimes prone to work an effect quite the reverse of that sought. It is important the residence should be in a clean, dry, airy locality, in a house that has no defects in sanitation, whether from plumbing, from decaying wood, or from water beneath the floors; to avoid the use of purgatives, especially of an acrid or drastic nature—the patent liver-pills, “pleasant” pellets and other nostrums, are extremely pernicious, the larger containing aloes, the smaller aloin, croton oil, or elaterium; to check any manifest laxity of the *prima viæ* by rest in the recumbent posture and employment of plain farinaceous foods; to use for drinking purposes only water that has been boiled, and when cold filtered through a mixture of sand and charcoal—I may state a personal preference for rain water filtered through the Kedzie filter, which I employed for many years.

If there is matter within the intestines tending to irritation, this should be gotten rid of by means of a simple aperient—Hunyadi Janos water, the effervescing draughts, castor oil, or the like; or if there are loose, watery evacuations, modification of the

secretions may be obtained by small and repeated doses of leptandrin, bismuth or cerium oxalate, ipecac, and camphor monobromide, combined; or by chloroform water or spirit; by coto; by chloranodyne; these may be employed either with or without aromatics. The tendency to intestinal flux, even if not choleraic, is necessarily a source of danger in that it must needs be more or less debilitating, thus inducing a condition that predisposes the individual to receive the cholera virus. It is obvious that conservation of the vital powers is of the utmost importance; but that an ordinary diarrhœa, the result of indiscretion in eating, of "taking cold," etc., can, *per se*, produce the specific germ of cholera, as some seem to imagine, is ridiculous to the extreme of absurdity.

But right here let it again be impressed that during a cholera epidemic the precise character of any diarrhœa must necessarily be difficult of determination; it may be truly choleraic, yet never manifest its real nature, and thus become a source of infection. For this reason, on such occasions all stools should be carefully gathered and cremated—no other measure is positively safe,—not emptied into the closet or privy; and these latter receptacles should be carefully disinfected. Again, no traveler or stranger should be permitted to use such closets—not even a neighbor; and no person should ever venture to enter a strange closet, privy, or latrine; since, as has already

been shown, the emanations therefrom may communicate the infection. No device of sanitary expert, or care of plumber, has yet sufficed to render a closet perfectly safe. The practice of wearing a broad, thick web of flannel as a belt, snugly embracing the abdomen, as a prophylactic against intestinal disturbances, far from being reprobated should be encouraged, especially among those of advanced life or sedentary habits.

Clothing soiled by diarrhœaic evacuations, whether the linen of the person or of the bed, should at least be subjected to the prolonged action of intensely hot water—be boiled, in fact,—and also of strong antiseptics, as measures of safety;* and if there is any suspicion of choleraic tendency, it is greatly to be preferred they too go to the fire. With all our boasted knowledge of antiseptics and antisepsis, we are as greatly in the dark as to what is available in destroying the poison of cholera, the element of fire excepted, as we are regarding the precise nature of the virus.

Finally, it may not be generally known that there is no disease in the whole nosological record that is more aided in the onset, and the fatal tendencies of

*I have already shown that Zehnder ascribed the origin of two cholera centres in Zürich in 1867, to an accumulation of bedding, mattresses, pillows, etc., which had been used on the beds of cholera patients, and which before disinfection were piled up in the neighborhood of the houses affected.—S.

which are more vigorously promoted and hastened by mental depression and fear, than cholera—less fear of cholera itself, however, than developed through other sources. The fact is, this malady in the main claims far fewer victims in proportion to the general population of any one country than many others that are regarded with comparative complaisance and often receive scarce passing attention save from medical men and those whose homes are directly invaded. Yearly whole districts are ravaged by typhoid fever, diphtheria, scarlatina, etc., to a greater degree than cholera is apt to do; further, I believe there are no valid reasons, other than the rapidity of the attack and the brief period that may elapse between inception and fatality, for regarding this disease, in fairly sanitary localities, as ultimately more dangerous, taking into consideration all results, than epidemic influenza. Again, it is notable that on every occasion when it assumes its most malignant and epidemic form, and spreads beyond the boundaries of the regions where it is endemic, there have been meteorologic or telluric conditions, or both, favorable to its dissemination; that generally throughout the temperate regions of the globe, as well as in the tropics, a marked tendency to enteric disease prevails*. In the present year (1892), as in 1891, this

* As early as the beginning of 1827 cholera appeared with renewed intensity at Calcutta, and here it is mentioned for the first time that many animals also showed the influ-

latter fact has been most manifest, and never for twenty-five years has the diarrhœaic mortality of London, Paris, Berlin, and other European cities reached greater height than during the past summer.

The present epidemic, as has been shown, readily found its way into Russia, where, by reason of the

ence of the disease (p. 365). . . . It was observed in North Germany at that time (1831) that chickens and pigeons, and in many instances fishes, perished in great numbers. (p. 359).—Ziemssen's "Cyclopædia of Practical Medicine," vol. i.—See Appendix A.

Even in India the development of cholera demands a medium degree of humidity of the soil and air. Great and protracted dryness, as well as excessive long-continued moisture of the soil, are alike unfavorable; therefore it is that in the hot regions of the East Indies, where dryness predominates and rainfalls are scanty, the cholera breaks out, as a rule, in the rainy season; while in the hot regions of lower Bengal, where wet weather predominates and rainfalls are abundant, the malady prevails in spring seasons which lack their usual rain. Great weight is attached to the monsoon season by Indian physicians, who for the most part in past years have been upholders of the miasmatic theory. Von Pettenkofer undoubtedly states the truth when he explains the influence of the monsoon by the saturation of the soil. Again, in Paris in 1849, after a wet spring, cholera reached an unusual degree of fatality in the first eight days of June, which were very warm; on the ninth, however, occurred a violent storm, when the number of new attacks diminished one-third as compared with the days of the preceding week. The same was true in the same year at Vienna, in Austria, and Christiania, in Denmark.—S.

famine and the consequent train of circumstances which make an entire population susceptible to additional scourge, it obtained firm foothold, and thence, in spite of sanitary barriers, reached the more civilized portions of Europe; the same circumstances, precisely, existed in Southern Ireland prior to and during the epidemic of 1834, in which locality the malady was exceptionally fatal until the famine was in great measure relieved, and wholesome food became the rule rather than the exception—thanks to the bounties of the world.

This with reference to the epidemic form of cholera; for it must be remembered that sporadic cholera like the poor, is “always with us” to greater or less extent, and constantly exists in the Levant, in Southern Europe along the Mediterranean, and occasionally appears as an endemic even on the borders of the German Ocean, and in the far interiors of Canada and the United States: The fact these sporadic cases are generally classed as aggravated cholera morbus, cholera nostras, English cholera, cholera infantum, etc., matters little, since they are pathologically one and the same, differing only in degree—an opinion that is upheld by the very highest authorities in India and Europe.* And I here insist, taking every feature

*See “Pathological Researches” of T. R. Lewis; London, 1888. “Nature and Treatment of Sporadic and Epidemic Cholera,” by Alex. Harkin, London, 1885. “On the Origin, Habits, and Diffusion of Cholera,” by Sir J. Fayrer, K. C. S. I., M. D., F. R. S.

into consideration, and giving all negative evidence due weight, that cholera is *cholera* wherever it occurs, and its epidemic prevalence and intensity are phases or accidents in its history. Surgeon-Majors D. D. Cunningham, and J. M. Cunningham, the latter the Health Commissioner of India, and many others who are very properly considered as expert authorities on Asiatic cholera, believe that cholera nostras is the same precise malady, "exhibited under conditions unfavorable for its perfect expression—that at times the conditions may and do become favorable, and then an epidemic results." The disease occurring in the deltas of the Ganges and Irawadi, in Moscow, Hamburg, London, New York or Winnipeg, is practically the same; and within a quarter of a century I have seen numerous cases in the Great Lake region of North America as serious as any that ever bore the name Asiatic, two that would have been pronounced *asphyctic* had the malady only been raging as an epidemic!

[Since the above was written, it has been my lot to see three other cases of cholera nostras (*morbus*) in Detroit, either of which was so virulent there would have been not the least hesitancy on the part of anyone viewing in pronouncing true Asiatic cholera, had there been the slightest chance of infectious origin. I may further add, the bacillus of Koch was specifically identified in the dejections of all three; also, that all were speedily relieved by inhibition of the sympathetic.]

Owing, presumably, to the utter failure of remedies, external or internal, to check the march of this dire disorder, the efforts of medical men for the most part—almost exclusively, I should say—have been directed to measures of prevention; and it is perhaps for this as well as for other reasons, that the therapeutics of cholera has not kept pace with that of other diseases. Sanitary measures have almost wholly superseded sanatory considerations, and to such an extent that in most dissertations of the leading advocates of State Medicine, the former are gravely heralded as panaceas for all epidemic disorders, and *sure*, in course of time, to eliminate from the nosological record the whole train of such accidents. Says Dr. Alex. Harkin:*

“These enthusiasts seem to have adopted for their motto, and emblazoned on their ‘banner with the strange device,’ *Sanitas Sanitatum, et omnia Sanitas*; and yet I fear the saying of the Wise Man, *Vanitas Vanitatum, et omnia Vanitas*, is not altogether obsolete in their regard, but fairly applicable to many of their most confident vaticinations. I am not one unfairly to decry the value of sanitation or scientific hygiene—I should rather prefer that sanitary and therapeutic measures should go hand-in-hand; but in the recent experience of a fatal form of typhoid fever, which numbered many victims in some

* Dublin Journal of Medical Sciences March, 1890.

of the finest cities of America, France, and Ireland, and in the actual presence of epidemic influenza which has prostrated thousands in the British Isles and on the continents of Europe and America, the impotence of sanitary arrangements to alone repel an attack of infectious disorder must be hopelessly apparent.”*

Looking at the history of cholera in Malta antecedent to 1887, we find after the epidemic of 1865, considered the most fatal up to that date on record, every measure that sanitary science, or engineering skill could effect was carried out; yet, notwithstanding, the cholera unheeding swept down upon the Island in 1887 in a more malignant form than ever, for out of 626 cases only 164 recovered—a death rate of 73.5 per cent. In 1865, after which Dr. Sutherland and the eminent engineer Osbert Chadwick visited the Island, the mortality stood at 60 per cent.; while in 1867, an intermediate visitation, there was a mortality of only 64 per cent., and this before the sanitary improvements were complete. In the presence of such adverse statistics it is difficult to agree wholly with sanitary scientists.

* This conviction appears to be entertained by some of the most enlightened organs of public opinion, as I find in an able article on Influenza in the Standard (London) this sentence: “And those who hoped that in the case of this, as of more serious scourges, the comparative excellence of sanitation would secure an absolute immunity, have to confess they have carried their faith in drainage a little too far!”

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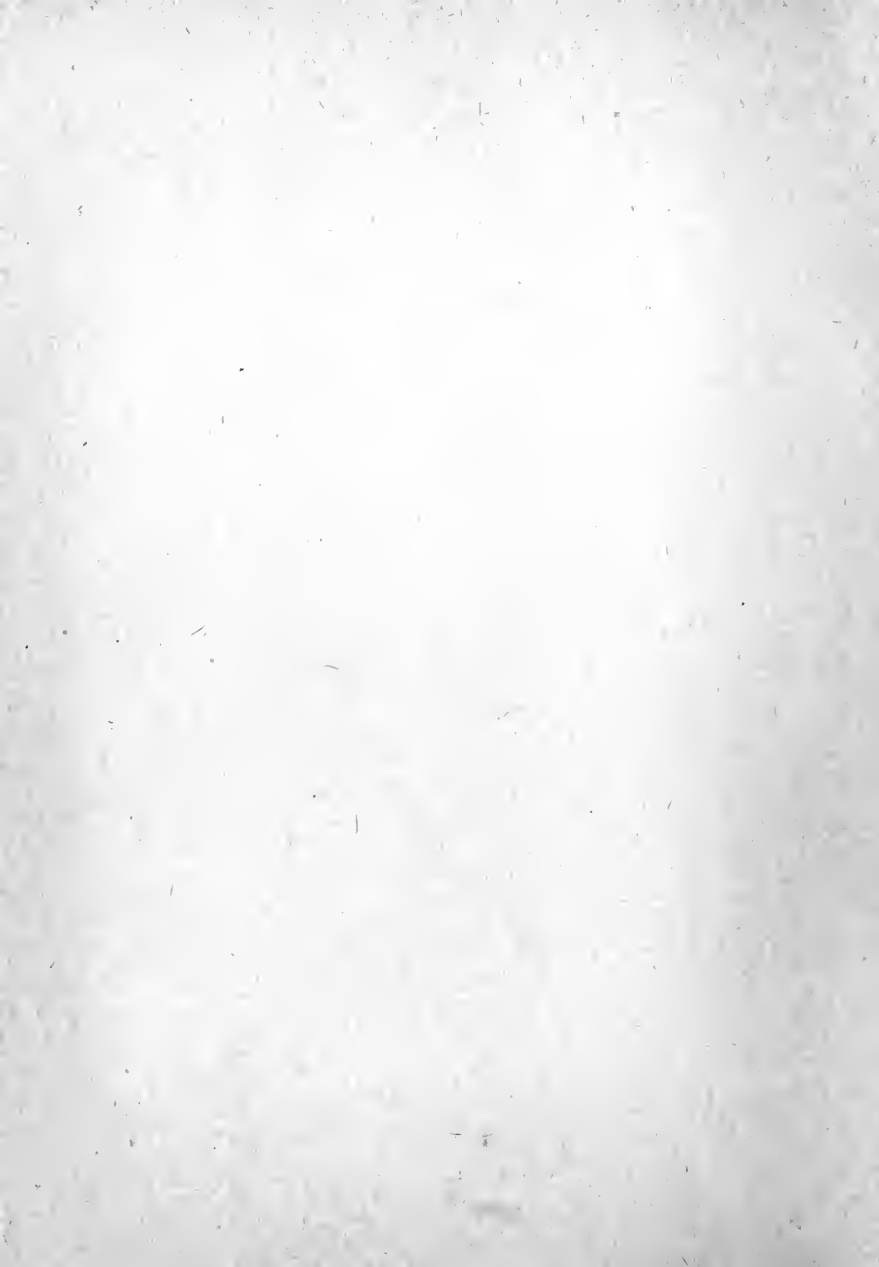
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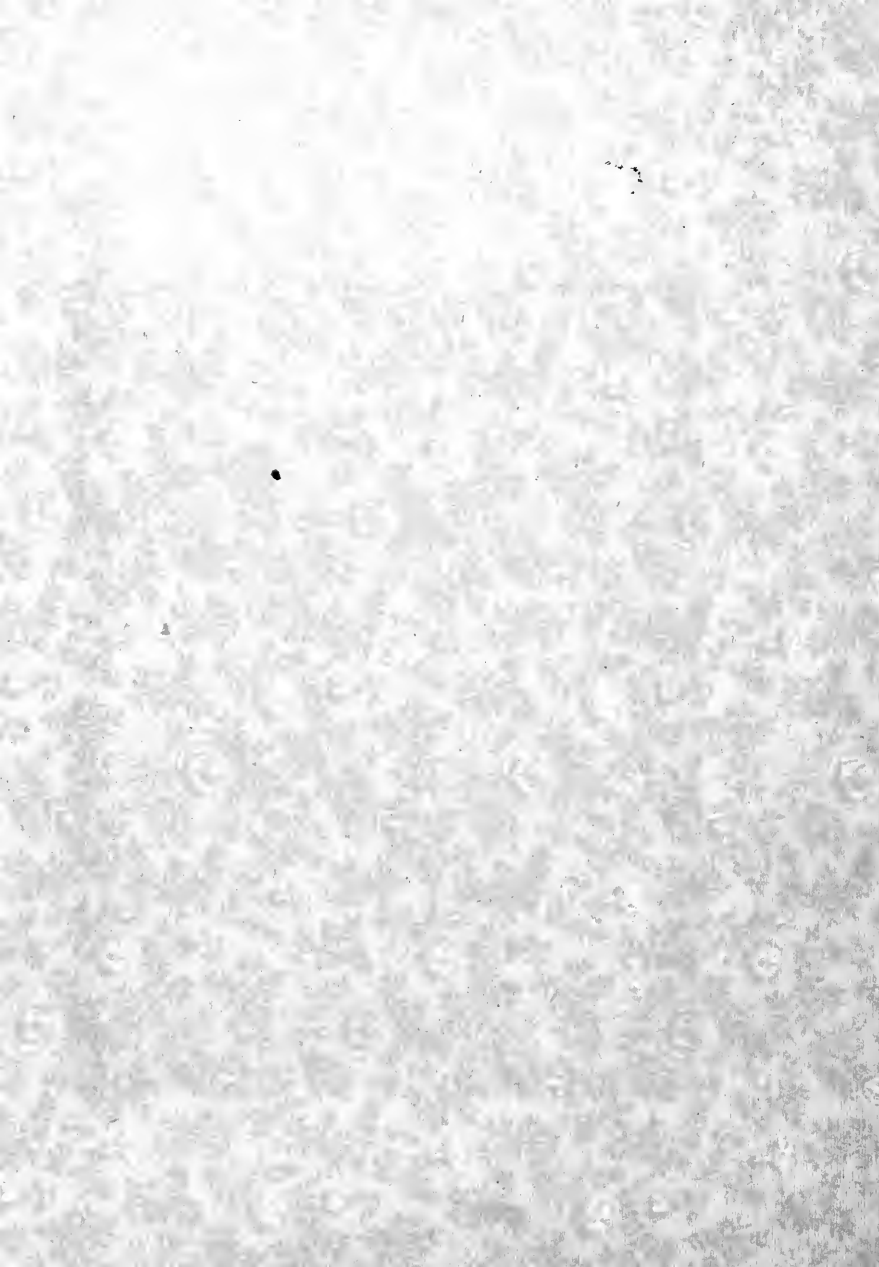
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